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TRANSACTIONS

AMERICAN ACADEMY
of
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JULY - AUGUST
1958

Volume 62 No. 4

ANNUAL MEETING, OCTOBER 12-17, 1958, CHICAGO

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1. *Cecil's Textbook of Medicine*, 7th ed., 1947, p. 1287.
2. *Ibid.*, p. 1598.
3. *Am. J. Ophth.* 42:771, 1956.
4. *Am. J. Digest. Dis.* 22:5, 1955.
5. *Med. Times* 84:741, 1956.

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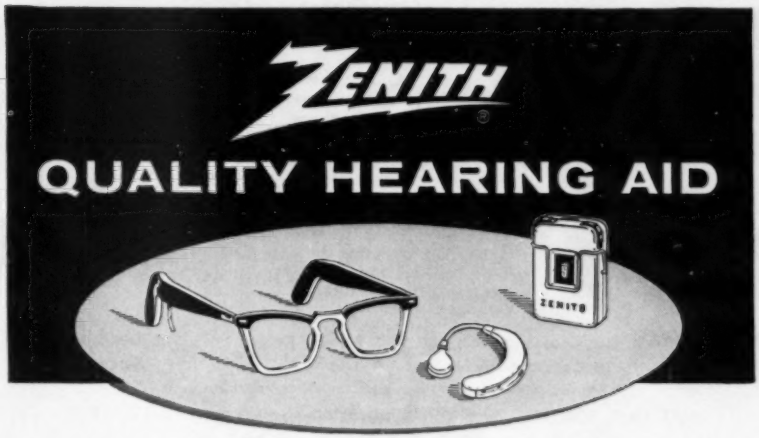

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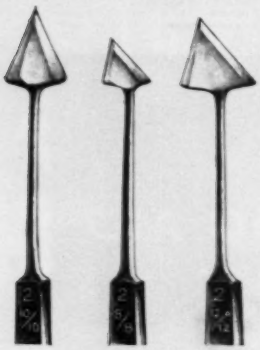

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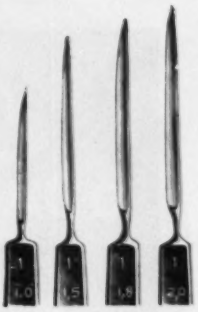

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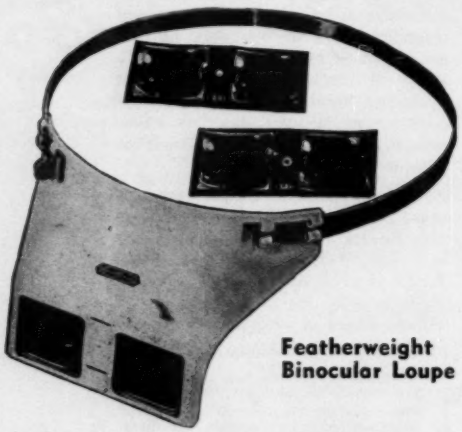

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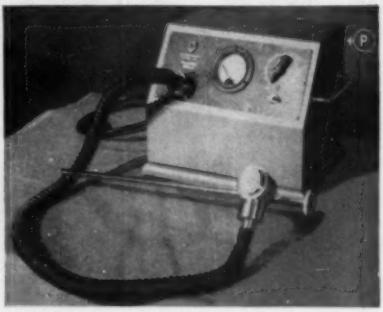

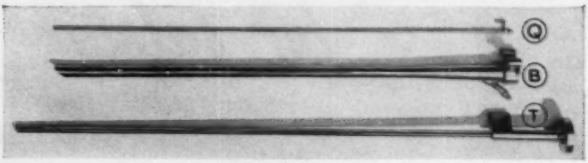

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Jackson, C.L.: New Instrumentarium for Peroral Endoscopy and Endoscopic Photography. LARYNGOSCOPE, *67*:777, (Aug.) 1957.

Fourestier, M., Gladu, A., and Vulmiere, J.: LA PRESSE MEDICALE, *60*:1292, (Oct.) 1952.

Dubois De Montreynaud, J.M., Edwards, Roger, J., and Gladu, Amédé J.: LARYNGOSCOPE, *66*:637, (June) 1956.

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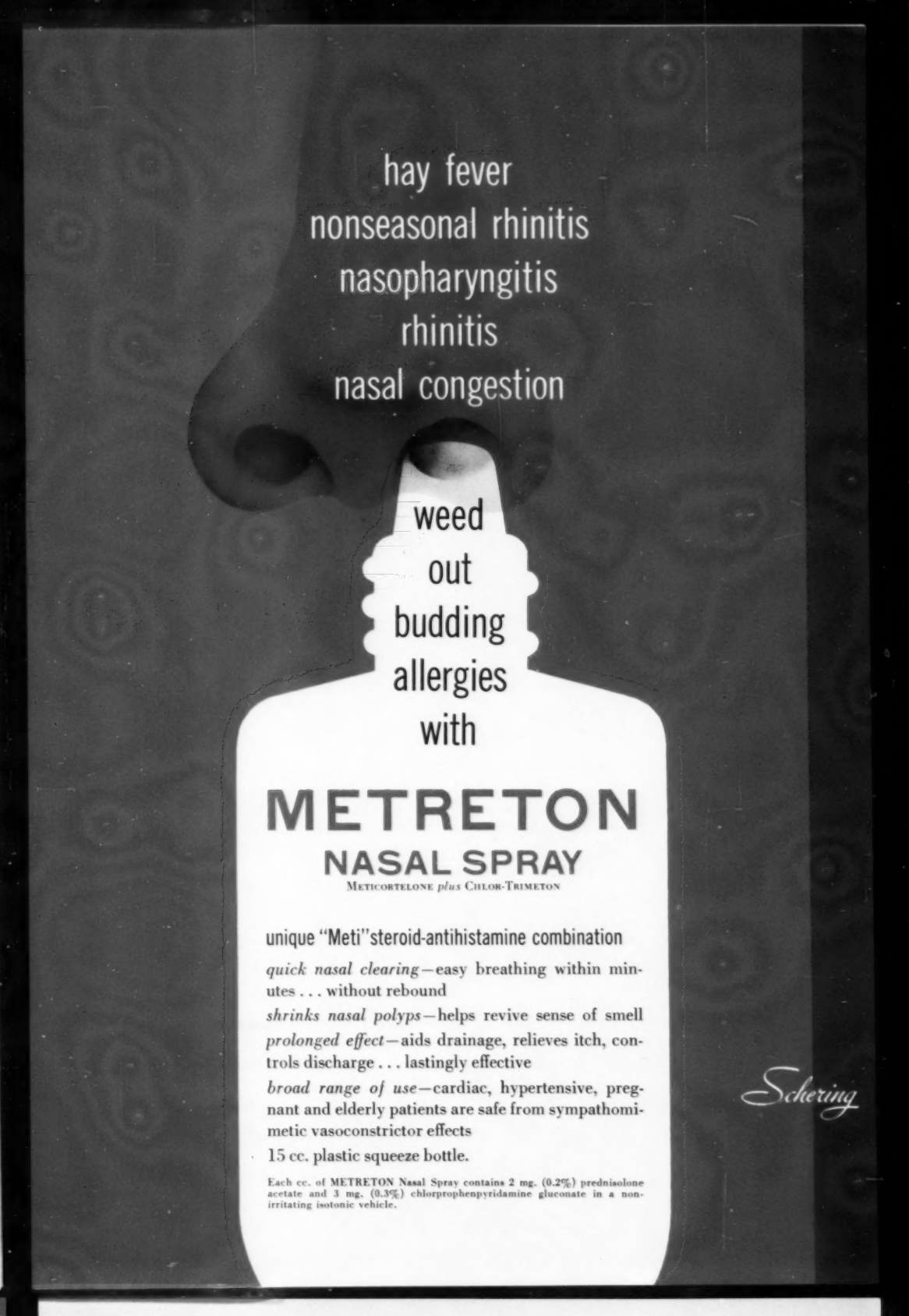
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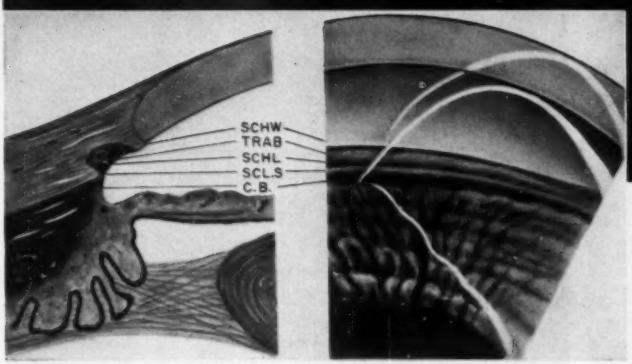

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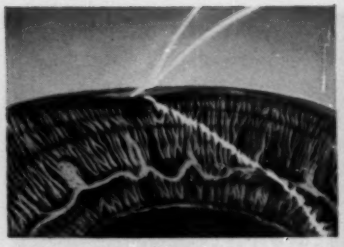

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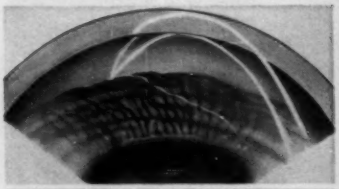

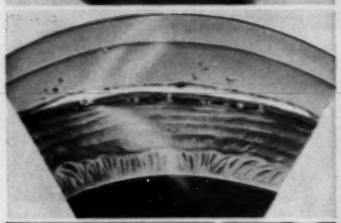

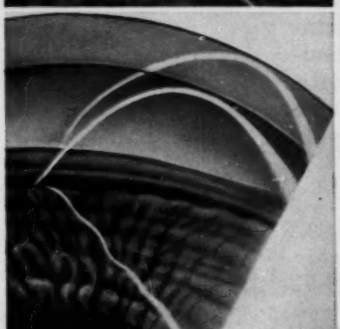

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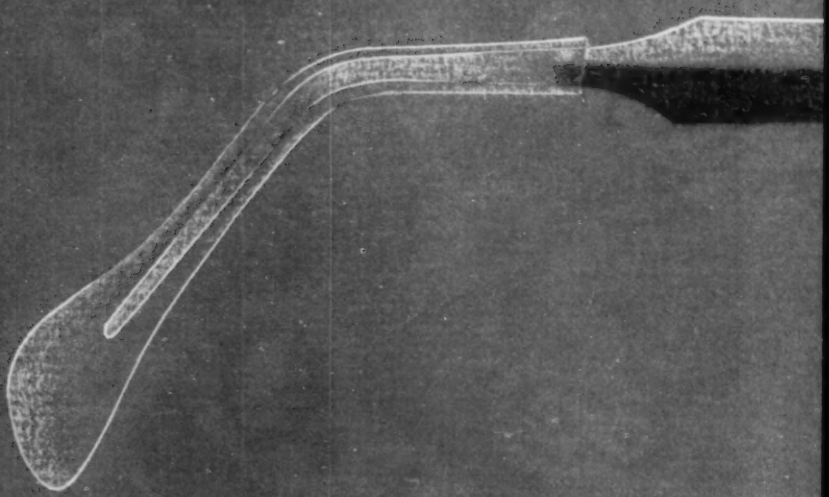

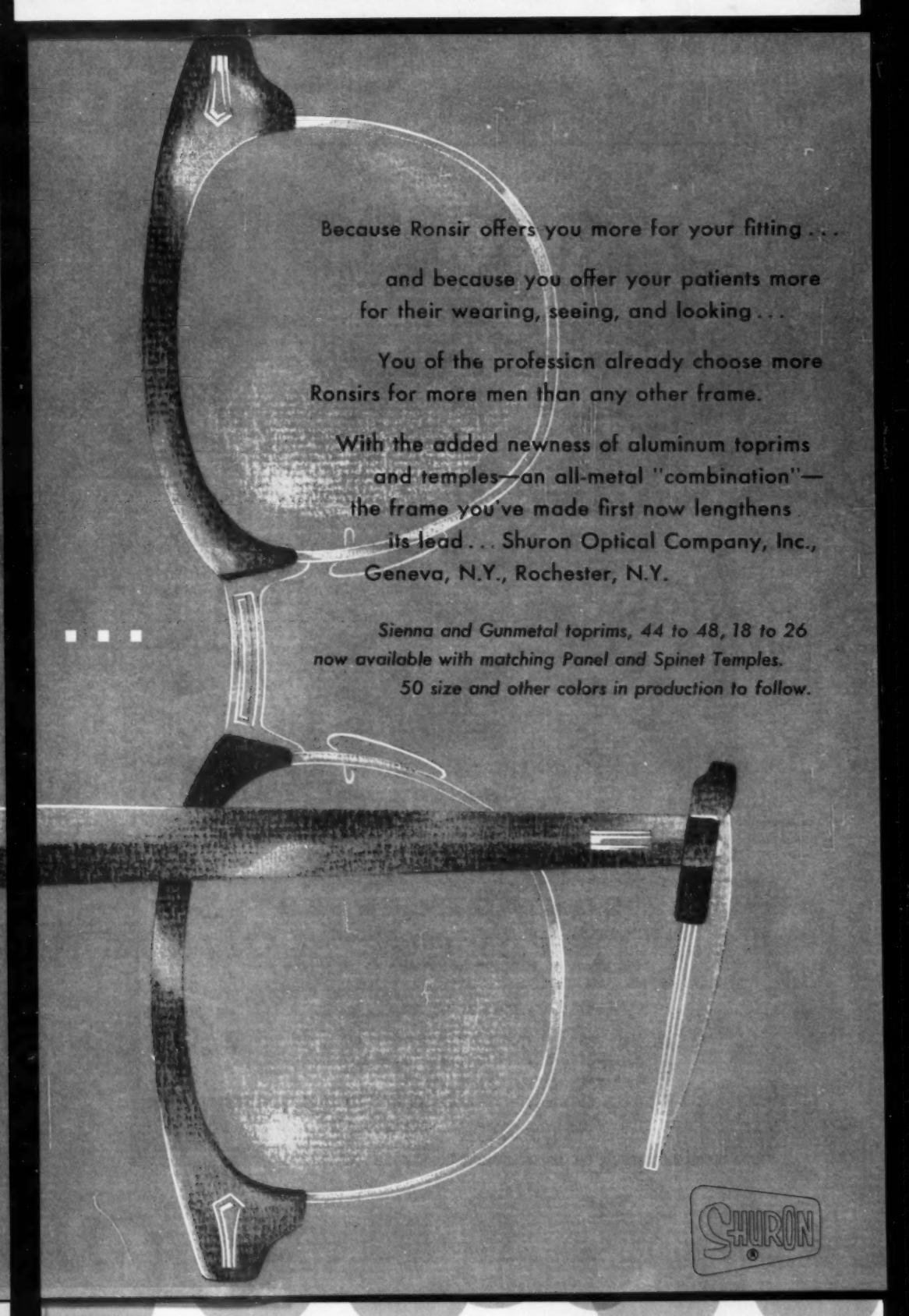
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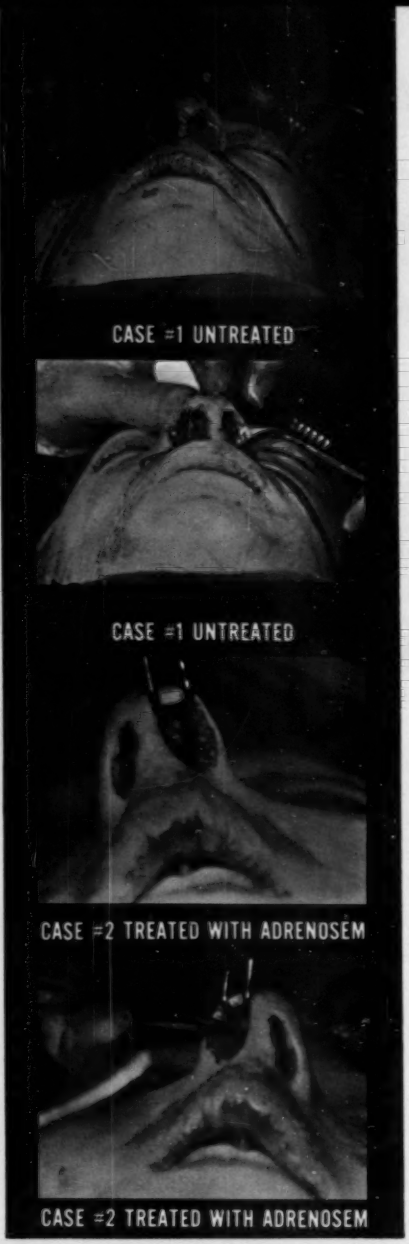

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1. Orzac, E.: Medical Care of the Child Patient Before and After Adenoidectomy and Tonsillectomy, N.Y. State J. Med. 56:886 (Mar., 1956).

2. Ersner, M.S. and Lerner, S.S.: M. Clin. North America 40:1749 (Nov., 1956).

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5. Brown, W.S.: The Use of Adrenosem Salicylate to Control Postoperative Bleeding in Plastic Surgery: Dermabrasion, Northwest Med. 57:470 (Apr., 1958)

6. Perkins, R.E.L.: A Clinical Investigation of Adrenochrome Monosemicarbazone Sodium Salicylate, Oral Surg., Oral Med., Oral Path. 10:230 (Mar., 1957).

7. Brode, A.B. and Chianese, T.C.: A Clinical Evaluation of Adrenosem Salicylate. Ann. of Dentistry 15:56 (Sept. 1956).

8. Zubieta C.B. and Escanaverino, R.B.: Adrenosem for the Prevention of Bleeding in Tonsillectomy, Am. Pract. 3:385 (Mar., 1957).

9. Riddle, A.C., Jr.: Adrenosem Salicylate: A Systemic Hemostat, Oral Surg., Oral Med., Oral Path. 6:617 (June, 1955).

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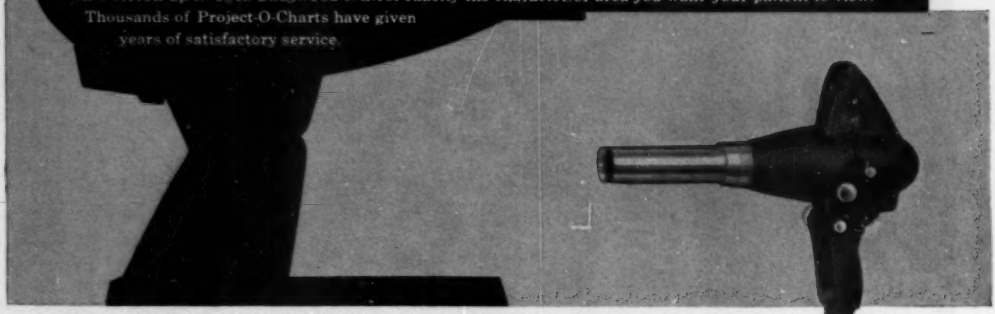

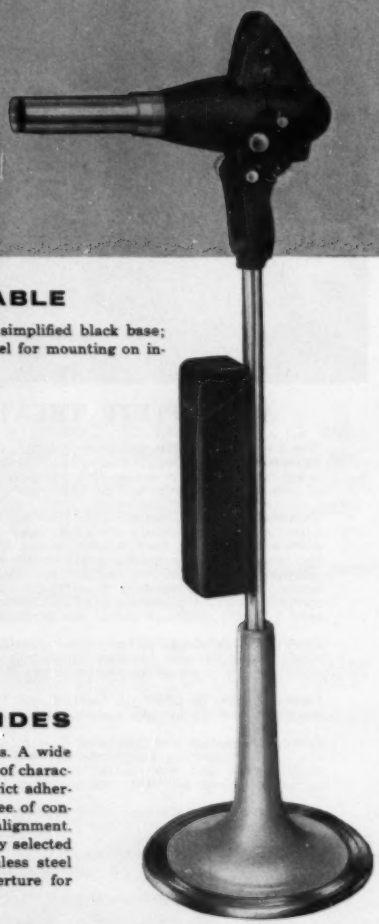

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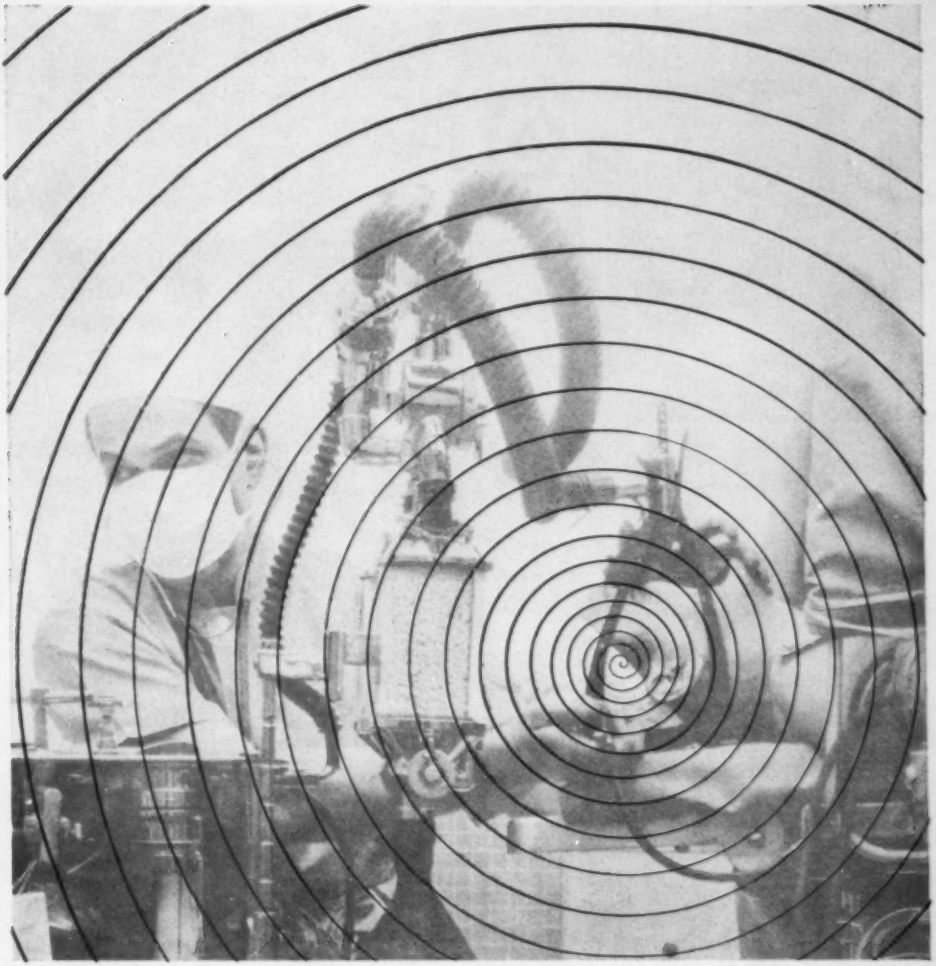

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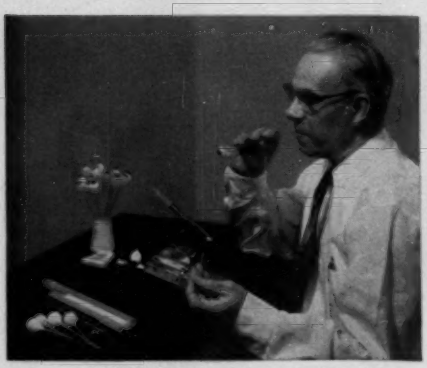

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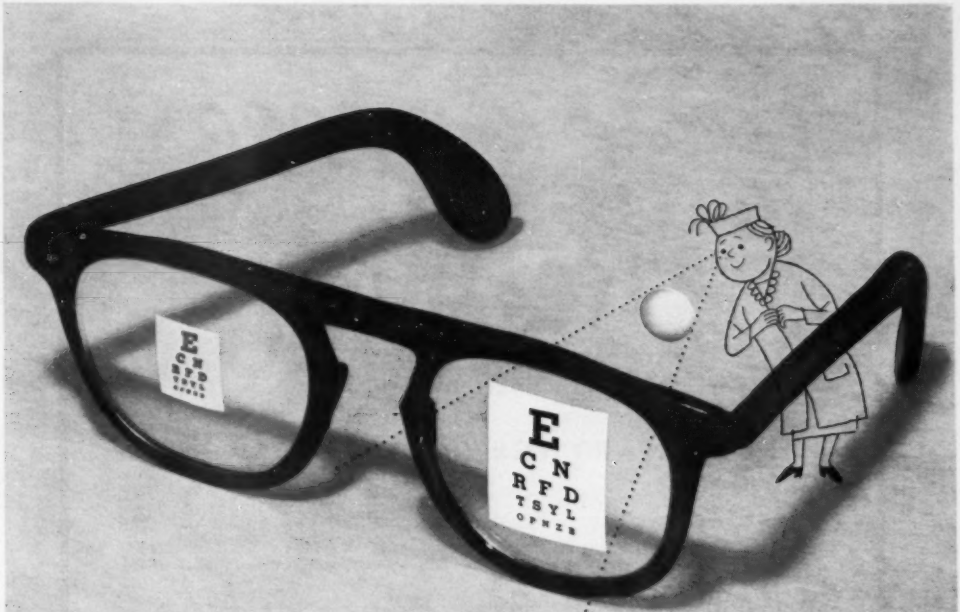

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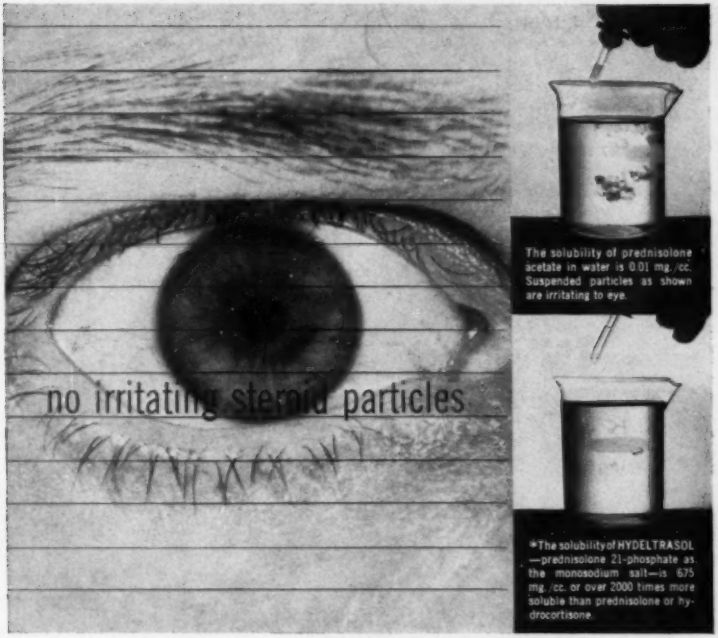
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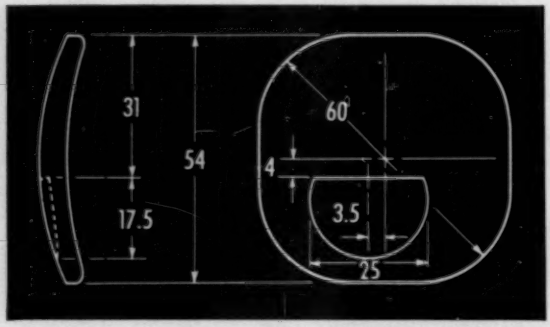

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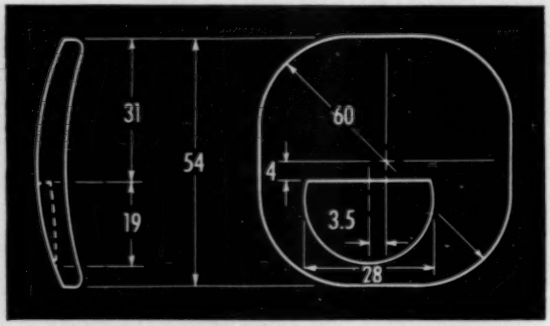

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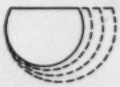

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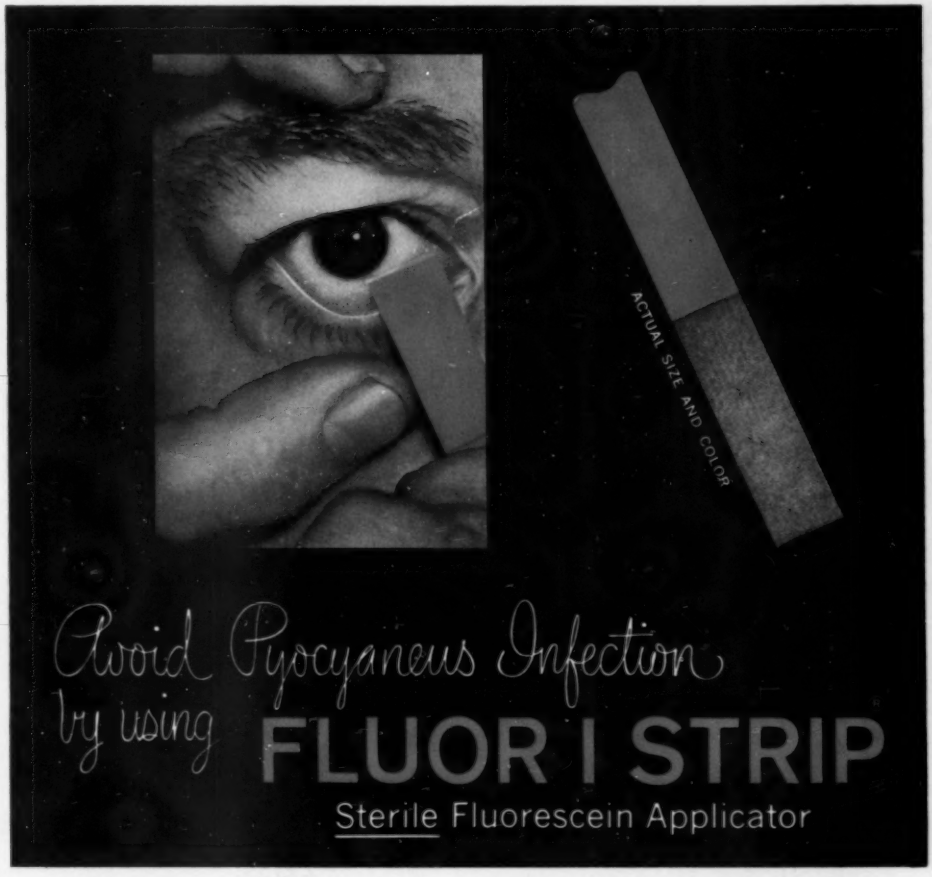

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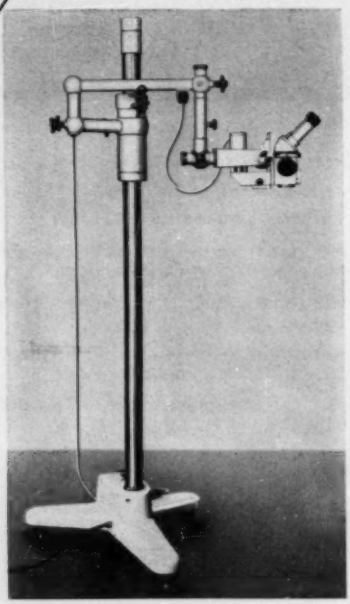

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THE DIAGNOSIS AND MANAGEMENT OF INTRAOCULAR MELANOMAS

A Symposium

Members of the Panel

EDWIN B. DUNPHY, M.D., *Moderator*
BOSTON, MASSACHUSETTS

ARNOLD W. FORREST, M.D.
WHITE PLAINS, NEW YORK

IRVING H. LEOPOLD, M.D.
PHILADELPHIA, PENNSYLVANIA

ALGERNON B. REESE, M.D.
NEW YORK, NEW YORK

LORENZ E. ZIMMERMAN, M.D.
WASHINGTON, D. C.

THIS symposium on intraocular melanomas will be conducted in a rather less formal manner than is the usual custom at American Academy meetings. It is not our purpose merely to cover standard procedures, which can be easily found in the textbooks, but rather to explore new ideas on the subject in the light of constantly increasing scientific knowledge. The members of this panel have had several meetings and have pooled their individual experiences and impressions so that they all are in essential agreement. While making no claim to dogmatism, it is hoped that this symposium will fill a need in answering some of the questions which often confront the practicing ophthalmologist, on whose shoulders rests the responsibility for proper diagnosis and management of intraocular malignant lesions.

Presented at the Sixty-Second Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 13-18, 1957, Chicago.

Although the diagnosis of an intraocular malignant lesion is frequently a simple matter, one always has some misgivings about enucleating the eyeball of a patient with good vision when biopsy has not been feasible. Every pathologic laboratory receives eyes, supposedly malignant, in which no tumor is found. Also, many eyes enucleated for other reasons are found to contain tumor where none was diagnosed preoperatively. Therefore, problems still exist as to diagnosis and, even after this has been made, there comes the question of proper management and prognosis. Let us first consider some of the diagnostic problems. Since there are no characteristic diagnostic symptoms of melanoma, the objective signs are all important. I am going to address my initial question to Dr. Forrest:

1. *Tell us whether there are any external signs which might suggest malig-*

nant melanoma of ciliary body or choroid.

DR. FORREST: Most intraocular tumors, in terms of external signs, are silent. The presence of external signs of malignant melanoma of the choroid or ciliary body is gratuitous.[38] It is helpful in indicating the existence of tumor and aiding in differential diagnosis. The absence of external signs is of no significance.

1. An important sign is an increase in number or a dilatation of blood vessels in the episclera, coursing over the globe in a quadrant or hemisphere occupied by the new growth. Rarely, extensive tumor necrosis may result in actual episcleritis or panophthalmitis.

2. An extraocular extension of the growth may be seen as a vascularized mass on the sclera or as a small nodule which may not be noticeable unless the patient is directed to look in extreme gazes with the lid retracted. If the growth has extended into the ciliary body, the extraocular nodule may appear at the external opening of the emissary through which the anterior ciliary artery courses through the sclera.

3. A blunt instrument may be used to palpate the sclera over the region of a malignant melanoma of the uveal tract when its location is sufficiently anterior to be accessible. Palpation by this means reveals that the sclera is more resistant over the site of the tumor. This sign is useful also in preparing globes in the pathology laboratory.

4. The presence of melanosis oculi is an additional supportive sign for the presence of tumor, indicating that tumor is a more likely diagnosis than inflammatory mass or idiopathic detachment. Although melanosis oculi itself is uncommon, an unusual number of these eyes contain tumors.

5. In large tumors, especially of the ciliary body, the pigmented mass may appear in the pupil.

6. Among the extremely rare external signs are the following: (a) Anesthesia of a sector of the cornea corresponding to the site of the lesion has been noted; this is presumably due to involvement of the ciliary nerve. (b) A partial paralysis of the iris may occur in an early stage of malignant melanoma of the choroid or ciliary body in the meridian in which the tumor is located. In this sector the iris remains less dilated even after atropine instillation. (c) Magnification may be necessary to identify dilated blood vessels of the iris, again in the area of iris corresponding to the tumor in the posterior segment. (d) Pigment dust may be noted over the surface of the iris as well as over the posterior surface of the cornea.

7. A melanoma of the ciliary body may present anteriorly through the iris or at an iridodialysis site and be visible externally. On occasion, cells of an anteriorly situated ciliary body tumor may be found implanted on the iris surface and growing as discrete lesions or extending from the angle.

Finally we would emphasize again the infrequency of these signs; they are not essential to diagnosis but are merely corroborative evidence.

2. *Dr. Reese, if the ophthalmoscope were the only diagnostic instrument you had at your disposal, how would you use it to the best advantage in diagnosing a malignant melanoma of the choroid?*

DR. REESE: The most valuable aid by far, in our opinion, is the inspection of the suspected lesion by retroillumination. This term does not refer to the usual transillumination. Retroillumina-

tion is employed, probably to some extent consciously or unconsciously, by most ophthalmologists. Its effectiveness, however, is greater if it is practiced with deliberation.

The test consists essentially of viewing the lesion through a well dilated pupil while a very bright ophthalmoscope light is thrown below its lower margin. It is important to move the head and the light until the reflected rays from the choroid reach the examiner's eye. Actually, the test cannot be carried out satisfactorily unless the light can be placed below the suspected lesion so that the observer's eye can receive the reflected rays above. Some manipulation of the light and the head may be required before reflected rays are seen, and then they may be observed only at one location. In subtle lesions, when this exact spot is determined the test may be most revealing. If the lesion is a melanoma, the reflected rays do not penetrate the lesion; therefore, its border is seen as an opaque or dark zone. Lesions simulating a melanoma transmit the reflected rays and therefore appear to be translucent.

Another useful sign is the appreciation of an abnormal vascular pattern over the surface of the lesion. This is seen as wide vascular channels of a nature and design foreign to the choroid of the retina. These blood sinuses represent varicose veins from passive congestion due to the constriction of that portion of the tumor which has extended through the elastic lamina vitrea (Zimmerman[56]).

Still another sign which may be of help is the occurrence of light brown pigmentation. This is in contrast to the jet-black pigment associated with proliferation of the retinal pigment epithelium. All acquired pigment from reparative and proliferative processes comes from the black pigment epithelium, because the melanoblasts of the choroid, which usually fabricate a light brown pigment, cannot reproduce their kind. As a melanoma comes from the melanoblasts of the choroid, the clinical appearance of light brown pigmentation favors a diagnosis of melanoma.

DR. DUNPHY: Another question for Dr. Reese:

3. *In your experience, what are the conditions which must be differentiated clinically from malignant melanoma of the choroid?*

DR. REESE: The answer to this question is based on the analysis by Ira S. Jones and me of 214 patients examined because a malignant melanoma of the choroid was suspected.[40] One hundred and six patients in this group did not have a malignant melanoma, as proved by the subsequent course of the disease, but had a lesion which simulated one. The pathologic conditions of this group of patients can be broken down into the following list in the order of their frequency:

Inflammation	39
Macular degeneration	21
Benign melanoma of the choroid	16
Metastatic carcinoma	9
Serous detachment of the retina	9
Benign melanoma of the retinal pigment epithelium	4
Hemorrhage	2
Hemangioma of the choroid	2
Coats's disease	1
Cyst of the macula	1
Congenital crater of the optic disc	1
Angioid streaks	1
Total	106

It can be seen that 60 per cent of these were inflammation or macular degeneration. These two groups will be discussed.

The lesions grouped under "Inflammation" were either active choroiditis, sequelae of choroiditis, or active effusive choroiditis with large bullous elevations of the retina. The lesions were suspect-

ed of being melanomas because of their dark color, which in 70 per cent of these cases was thought to be caused by the stimulation of the pigment epithelium to proliferate. In the other instances, the dark color seemed to be due to deeplying hemorrhage which may have been converted, to some degree, to hemosiderin.

Fifty per cent of the lesions diagnosed as inflammation showed residues of edema or inspissated edema at some site in or around the lesion. Such a finding, in our experience, is never seen accompanying a melanoma.

In inflammatory lesions of the fundus in the active phase, hemorrhage is noted in approximately 50 per cent of the cases. The characteristic feature of the hemorrhage is that it is prone to occur around the lesion as a collarette. In melanomas, hemorrhage plays a most inconspicuous part, especially in small tumors. Vitreous hemorrhages are very rare indeed—three out of 84 eyes analyzed—and they accompany the larger lesions which are either necrotic or have broken through the lamina vitrea, which produces a constriction around the neck and causes varicosity of the veins from passive congestion.

Twenty-five per cent of the inflammatory lesions showed some indication of a similar process in the fellow eye.

Approximately 50 per cent of the cases of macular degeneration which belong to the disciform type showed some associated edema residue, either in or around the lesion. One-third showed sufficient pathologic changes in the macular area of the fellow eye to give confirmatory evidence.

Too much emphasis cannot be placed on the importance of examining the fellow eye. Frequently pathologic change is found here which is of the greatest importance in arriving at the correct diagnosis—25 per cent of suspected inflammatory lesions and 33.3 per cent of macular lesions show some indication of a similar process in the fellow eye.

4. *Dr. Zimmerman, as a pathologist will you show us some of the other lesions you have found in eyes enucleated because of an erroneous diagnosis of malignant melanoma?*

DR. ZIMMERMAN: Many of the eyes we receive have been enucleated because they were blind and painful. The combination of retinal detachment and secondary glaucoma in eyes that cannot be examined satisfactorily because of opaque media often arouses suspicion of a hidden tumor. It is not surprising, therefore, that we frequently find "possible intraocular tumor" appended to other clinical diagnoses. However, this is not the type of diagnostic problem that I have selected for presentation here. Rather, I have selected cases in which definite lesions visible through clear media simulated melanomas so closely that enucleation was performed.

Non-neoplastic proliferative lesions of the macular region (Kuhnt-Junius or disciform degeneration) constitute one of the more important groups of lesions in eyes enucleated because a melanoma is believed to be present. A few years ago William C. Frayer[19] reviewed 23 cases of this kind in the Registry of Ophthalmic Pathology of the Armed Forces Institute of Pathology. In 12 of these (52 per cent) the eye had been enucleated because of the diagnosis of melanoma, while in the remainder the macular lesions were incidental observations in eyes enucleated for other reasons (glaucoma in 10 of the 11 cases).

The following history is from one of several cases received since the completion of Frayer's study:

AFIP Acc. 685300. A 58-year-old white man complained of blurring of vision of the left eye which had increased progressively

since its onset two years earlier. Examination revealed an elevated area measuring 3 × 4 disc diameters in and about the macula. It was surrounded by yellowish gray lesions similar to those characteristic of circinate retinopathy. A scotoma corresponding in location to this macular lesion was present. Visual acuity was 20/70. The tension was normal. Funduscopic examination of the other eye revealed only Grade I arteriosclerosis. A complete radiologic survey yielded no evidence of metastasis, and the urine was negative for melanin. The left eye was enucleated because the lesion was considered a "possible intraocular tumor."

Examination of the specimen received in the laboratory revealed a swollen macula and a small (5 mm. in diameter) flat retinal detachment below and temporal to the macula. Many minute yellowish granules were seen over the surface of this part of the retina. Microscopically the characteristic exudative and proliferative features of disciform degeneration were present. Between the disc and the macula, serous exudate separated the retinal pigment epithelium from the elastic component of Bruch's membrane (fig. 4-1). In places the exudate was partially organized by fibroblastic and vascular proliferation, and some areas were already densely fibrous (fig. 4-2). Mild proliferation of the pigment epithelium was noted. The outer retinal layers in the macular region had undergone extensive degeneration but elsewhere the retinal architecture was preserved.

Hemorrhagic detachments of the retina and choroid were among the most frequent causes for the erroneous interpretation of malignant melanoma in the study made by Harold Q. Kirk and Robert W. Petty.[26] The following case from the Registry is typical:

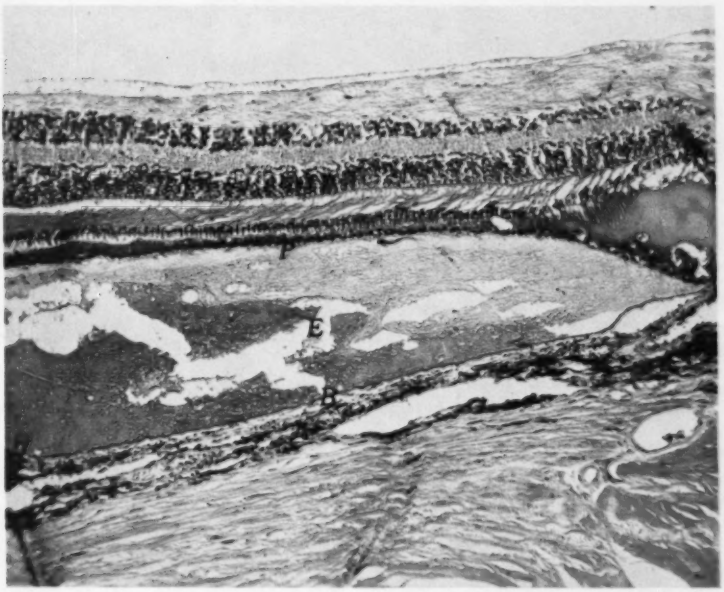

FIG. 4-1—AFIP Acc. 685300. Disciform degeneration of macula. Serous exudate (E) separates the retinal pigment epithelium (P) from Bruch's membrane (B). There is extensive degeneration of the layer of rods and cones and of their nuclei. (Masson trichrome stain, × 90)

AFIP Acc. 595574. A 53-year-old woman was first seen in April 1953 because of blurred vision and flashing lights in her right eye. Visual acuity was 20/40. A grayish brown mass, elevated two diopters, was seen beneath the retina between the disc and the macula.

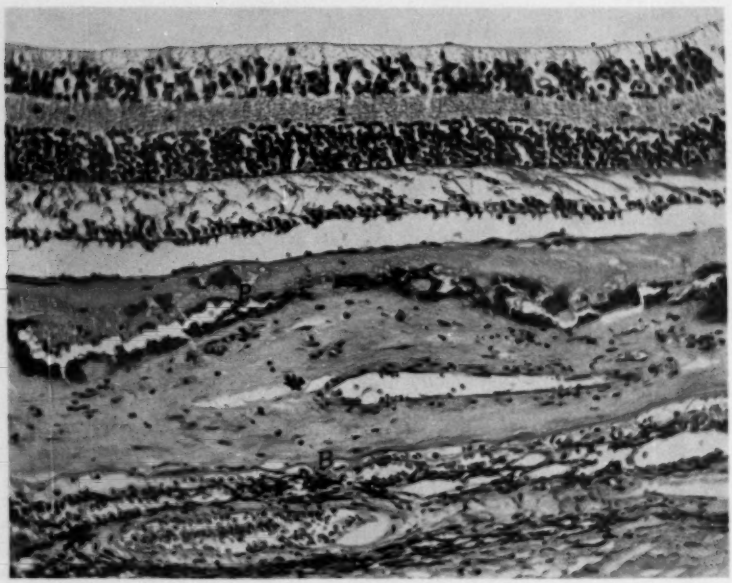

FIG. 4-2—Another portion of the macular region from same eye illustrated in Fig. 4-1. Here there is hyperplasia of the pigment epithelium (P) and a thick plaque of fibrovascular tissue separates the pigment epithelium from Bruch's membrane (B). (H & E, × 140)

Definite enlargement was demonstrated by serial photography, and the eye was enucleated. Histopathologic study revealed recent submacular hemorrhage. A layer of blood had dissected between the pigment epithelium and the lamina vitrea to form a discrete hematoma (fig. 4-3). The patient was seen by another ophthalmologist in December 1955 because of a similar lesion in her remaining eye. He wrote for confirmation of the histopathologic diagnosis in the enucleated right eye, because it would "influence the decision on the handling of the present condition."

Serous separations of the retina, particularly if of long duration and associated with glaucoma, may be mistaken for malignant melanoma. Following operations for retinal detachment the uptake of radioactive phosphorus may be significantly increased. This, together with the simulated retinal elevation produced by postoperative scleral flattening, may further confuse the picture and prompt enucleation, as it did in the following case:

AFIP Acc. 743282. A 22-year-old white man complained that vision in his left eye had been poor for ten years. There was no history of injury, but an operation for retinal detachment had been performed at another hospital in July 1955. Six months later the possibility of malignant melanoma was considered and the patient was transferred to a medical center where radioactive phosphorus uptake studies could be made. Examination revealed visual acuity reduced to perception of hand movements and a localized area of retinal detachment inferiorly. Pigment deposits were noted in this region. In March 1956 the patient was given an intravenous injection of 500 microcuries of radioactive phosphorus. Counts obtained one hour and twenty-four hours later demonstrated increased uptake localized to the lower temporal quadrant; this was interpreted as a significant indication of intraocular neoplasm.

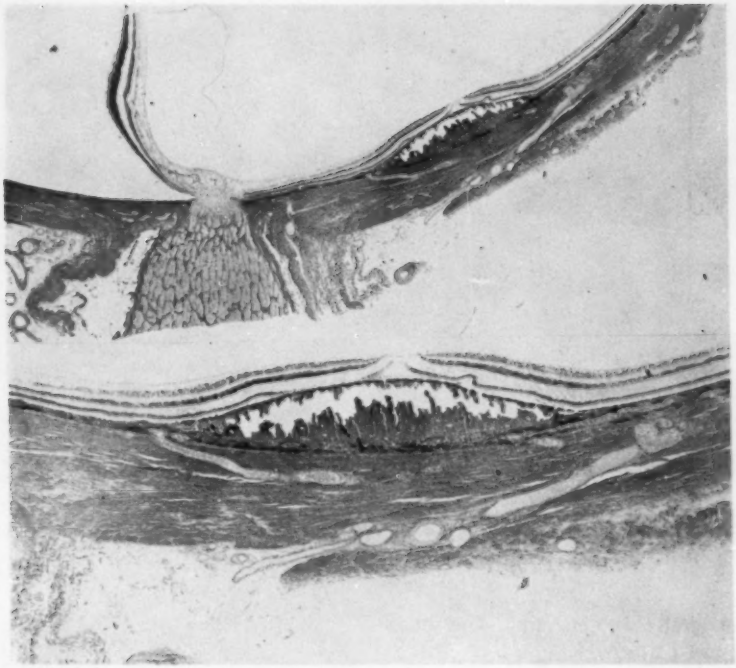

FIG. 4-3—AFIP Acc. 595574. Submacular hemorrhage. A layer of fresh blood has dissected the pigment epithelium away from Bruch's membrane. (H & E; Top, × 10; Bottom, × 22)

Examination of the enucleated eye revealed extensive flattening along the equator in the temporal and inferior quadrants, and thickening of the episclera at the site of the operation for retinal detachment. Increased lucency on this area was noted on transillumination. Upon opening the eye no tumor was found. There was extensive chorioretinal degeneration along the flattened portion of the globe (fig. 4-4).

Microscopic examination of the region thought to be the site of neoplasm revealed negligible inflammatory reaction and only slight fibroblastic proliferation in the irregularly thickened sclera and episclera (fig. 4-5).

Serous separation of the choroid and/or ciliary body is believed to occur commonly after cataract extraction. The condition seldom persists, but when it does it may be mistaken for a melanoma, particularly when the detachment re-

FIG. 4-4—AFIP Acc. 743282. Chorioretinal and scleral scarring following an operation for retinal detachment. Photomicrographs of the lesion between the arrows are shown in figure 4-5.

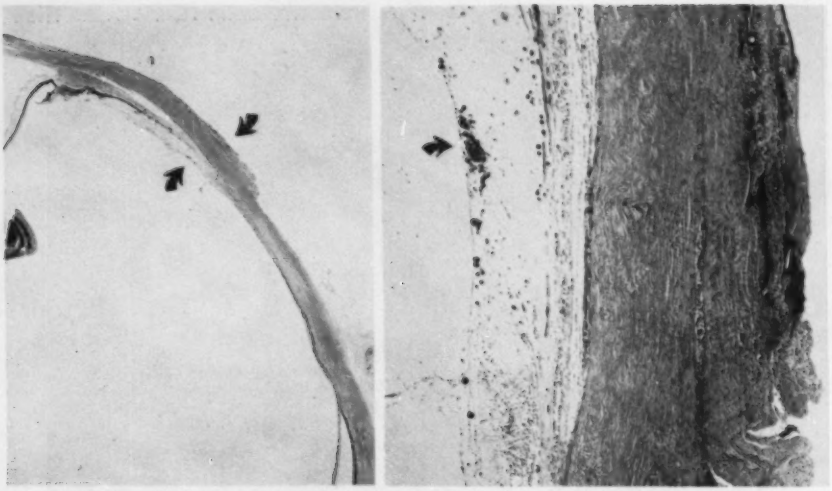

FIG. 4-5—Photomicrographs of gross specimen illustrated in figure 4-4. (Left) Extensive necrosis of retina, choroid, and ciliary body; irregular thickening of the scarred sclera and episclera. (H & E, × 6) (Right) Macrophages filled with pigment (arrow) are scattered in the necrotic retina. Very little active inflammation is observed. (H & E, × 115)

mains localized to one area. The following case is illustrative:

AFIP Acc. 680313. Combined extracapsular cataract extraction was performed on a 78-year-old white man on October 8, 1954. One week later a large pigmented mass which filled the nasal half of the interior of the globe was first noted. Visual acuity was 20/40 and the tension was 26 mm. Hg. The mass was interpreted as malignant melanoma and the eye was enucleated October 30.

Transillumination of the specimen in the laboratory revealed no shadows, but when the eye was opened extensive choroidal detachment was observed (fig. 4-6). There was no tumor.

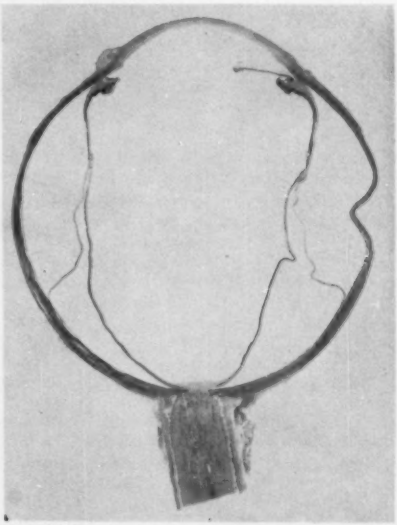

FIG. 4-6—AFIP Acc. 680313. Postoperative detachment of choroid and ciliary body. (H & E, × 3.5)

Choroidal detachment may also occur following glaucoma operations, and when the surgeon has not been able to see clearly into the eye before the operation, he may believe that melanoma has produced secondary glaucoma. Only rarely does the ophthalmologist enucleate an eye because he confuses choroidal detachment of obscure origin

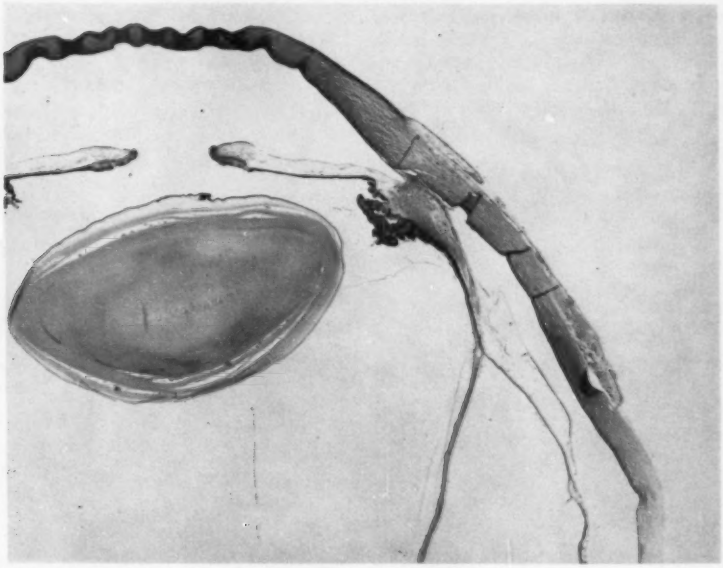

FIG. 4-7—AFIP Acc. 753400. Detachment of choroid and ciliary body presumed to be secondary to diffuse sclerosis of uveal blood vessels. Periodic acid-Schiff reaction. (× 8)

with malignant melanoma, but the following case shows that it can occur:

AFIP Acc. 753400. A 75-year-old white woman with a history of "three slight strokes" was seen because of swelling of the lids, chemosis, proptosis, and temporal displacement of her right eye. Vision in this eye was reduced to light perception. The tension was said to be normal (tactile). After dilatation of the pupil, a dark brown mass could be seen arising from the ciliary body and choroid on the nasal side. The mass formed a sharp ridge along the equator in the upper nasal quadrant. The temporal periphery appeared normal. The examiner's impression was that a large malignant melanoma had either caused gross perforation of the globe or produced orbital metastasis. The eye was enucleated two days later.

Transillumination of the soft globe in the laboratory revealed no evidence of tumor. The most significant histopathologic observation was severe diffuse sclerosis of the choroidal blood vessels of all sizes, from the larger ciliary arteries to the choriocapillaris, which had resulted in widespread degeneration of the outer retinal layers. The pars plana of the ciliary body and the peripheral choroid were separated from the sclera (fig. 4-7) accounting for the mass observed clinically. Our clinicopathologic interpretation was that the severe choroidal vascular disease had produced hypotony, which in turn led to uveal detachment.

Granulomatous lesions within the eye may lack the characteristic clinical features of "granulomatous uveitis" and, when localized, simulate melanoma. In a number of Helenor C. Wilder's cases of toxoplasmosis, the eye was enucleated because of a suspected tumor.[54] The following case is from her original series:

AFIP Acc. 120510. A 27-year-old white man complained of severe reduction of vision in his right eye and associated right-sided headache of six months' duration. Examination revealed what was interpreted to be retinal detachment with an underlying "melanosarcoma." When the eye was opened after enucleation, no tumor was seen, only a pale thickened chorioretinal lesion just above the macula. Microscopically, this was first con-

sidered a tuberculous granuloma (fig. 4-8), although acid-fast organisms could not be identified. Later Mrs. Wilder demonstrated parasites, morphologically identical with Toxoplasma, within the lesion.

scar secondary to an old penetrating wound. However, there was no confirmatory history of old injury.

Other tumors of the choroid, especially hemangiomas and metastatic car-

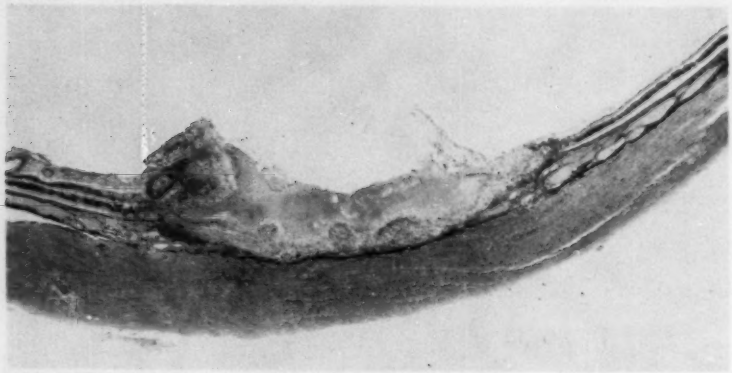

FIG. 4-8—AFIP Acc. 120510. Granulomatous chorioretinitis due to toxoplasmosis. (H & E, × 16)

Staphylomas are rarely confused with melanoma, but I have seen examples which simulated uveal tumors in both the anterior and posterior segments. First I will show you a small focal staphyloma of the sclera that was the cause for enucleation; later I will show you another case in which a similar lesion in the cornea was believed to be an iris tumor.

AFIP Acc. 633196. A 76-year-old white woman who had refused cataract extraction in 1949, complained of symptoms of glaucoma in her cataractous eye in 1952. When she was seen again in 1954, a black mass protruded from the sclera just anterior to the medial rectus muscle. Malignant melanoma was suspected and immediate enucleation advised. Examination of the enucleated eye in the laboratory revealed a small, crater-like excavation in the pars plana adjacent to the insertion of the medial rectus muscle (fig. 4-9). Uveal pigment lined the defect, which after microscopic examination we assumed to be a cystoid

cinomas, have often been regarded as melanomas until histopathologic study disclosed their true nature. It should be emphasized that metastatic carcinoma is not always the flat diffuse mass described in textbooks. In my experience a mass of collar button or mushroom shape (fig. 4-10) has not been at all rare. William Hart,[22] who has recently completed a review of cases of metastatic carcinoma in the Registry, found that while the percentage of correct clinical diagnoses of ocular metastasis from carcinoma of the breast is high, other metastatic carcinomas are usually mistaken for malignant melanomas. Most breast cancers metastasize to the eye late in the disease when other manifestations of carcinomatosis may be recognized; with other tumors, such as bronchogenic carcinoma, the ocular symptoms are more likely to be the patient's presenting complaint.

FIG. 4-9—AFIP Acc. 633196. Focal, crater-like staphyloma of sclera in region of pars plana, presumed to be secondary to an old penetrating wound. (H & E, × 50)

FIG. 4-10—AFIP Acc. 213695. Metastatic chorionepithelioma of choroid. The tumor has broken through Bruch's membrane to form a subretinal mass, simulating a primary malignant melanoma. (H & E, × 2)

Hemangioma of the choroid is frequently misinterpreted as malignant melanoma (fig. 4-11). Through the kindness of Dr. J. Jack Stokes,[46] who is making an extensive study of ocular hemangiomas, I can give you some of his data. In 21 of the 27 cases he has studied, the clinical diagnosis was malignant melanoma. The fundus was visible at the time of enucleation in 13 of the 27 cases, and in 10 of these the choroidal lesion was believed to be a malignant melanoma.

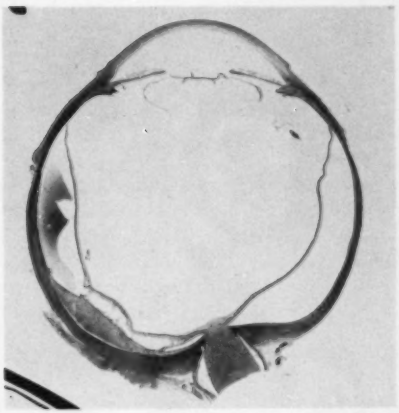

FIG. 4-11—AFIP Acc. 308619. Hemangioma of choroid seen clinically as a black pigmented patch, one-half disc diameter in size, located in the macular region. The patient, a 25-year-old white man, had noted failing vision for four years but the intraocular pressure was not elevated. A clinical diagnosis of malignant melanoma was made and the eye was enucleated.

DR. DUNPHY: From what Dr. Reese and Dr. Zimmerman have told us, it becomes apparent that every effort must be made to be sure of our diagnosis before subjecting an eye to enucleation. We must not neglect the various diagnostic aids at our disposal.

5. *Dr. Leopold, what is the value of transillumination in the diagnosis of suspected choroidal melanoma?*

DR. LEOPOLD: The two most widely used methods of objective transillumination are the transcleral and the transpupillary approaches. Usually, the transcleral method is employed: a light source is placed on the scleral surface behind the suspected area and the general illumination of the pupillary area is observed. The point of light must be behind the lesion to be transilluminated. In posterior lesions this will necessitate making a cut in the conjunctiva or Tenon's capsule and inserting the special instrument, such as that designed by Lancaster, directly behind the suspected lesion. The amount of illumination is compared with that obtained when the light source is placed in other areas uninvolved by the lesion to be diagnosed.

One can also observe the fundus with an ophthalmoscope, the source of illumination being the transilluminating instrument held over the sclera in the region of the lesion. This was originally suggested by Lindahl in 1920 and was further studied by Burke in 1936.

The transpupillary method requires a light source to be directed through the pupil. One notes the amount of light which emerges through the sclera.

Several variations of these methods have been reported. In addition, subjective transillumination can be employed in which the patient will note a sensation of reduced light or no light as the source of illumination is passed over the sclera at the site of the lesion.

All of these methods depend on the fact that a solid lesion will interfere with light transmission. Unfortunately, the findings through transillumination are not always clear-cut. Two careful observers may interpret the results differently, and there are conditions other than tumors which will cause a reduction in transillumination of light. Heavily pigmented eyes may interfere with transillumination. Hemorrhage and perhaps dense inflammatory collections may obstruct the light. On the other hand, there are tumors which will not interfere with light transmission, such as very small ones or a nonpigmented one. Transillumination is not a reliable diagnostic aid for intraocular malignant lesions.

6. *Is it of any value in iris tumors?*

DR. LEOPOLD: In transillumination of ciliary body and iris, the transcleral or the transpupillary method may be employed. Scleral illumination will also allow retroillumination of the iris. The observations may be made by the naked eye or with a loupe, ophthalmoscope, slit lamp, or gonioscope. One encounters the same difficulties with false positives and false negatives in dealing with lesions of the iris and ciliary body as encountered in questionable lesions of the choroid. Transillumination may be helpful because a cyst will transmit light whereas a melanoma will not, but a small tumor may be masked by an adjacent large clear cyst. Scheie has pointed out the value of gonioscopy following wide dilatation of the pupil for the diagnosis of iris cysts. Scleral illumination for the diagnosis of iris cysts may be obtained by a Barkan hand lamp and the observations made with the microscope head of a slit lamp for magnification. The light and one's line of gaze must be directed almost parallel to the lens-iris diaphragm to see the posterior chamber. However, some of the black pigmented cysts may not transmit light readily. Therefore, positive or successful transillumination is helpful but does not exclude the possibility of a small tumor with an associated cyst, whereas the failure to transilluminate does not necessarily indicate the presence of a neoplasm.

7. Dr. Reese, do you consider perimetry helpful in differentiating between a benign and a malignant melanoma of the choroid?

DR. REESE: Characteristically, a benign choroidal melanoma is evidenced by no field defect. In very rare instances I have been able to elicit a scotoma. It was smaller than would be expected from the size of the tumor, whereas the field defect from a malignant melanoma is larger than would be expected.

8. Dr. Leopold, you have had considerable experience with the P^{32} test in these lesions. Would you tell us how you feel about it as a diagnostic aid; and how should the test be performed to eliminate false positives and false negatives?

DR. LEOPOLD: Since the first report by Thomas, Krohmer and Storaasli[48] in 1952, many excellent studies have confirmed the fact that the measurable uptake of radioactive phosphorus is greater over malignant melanomas of the eye than over normal tissue, intraocular hemorrhage or serous retinal detachments. (See references 4, 11, 13-16, 25, 35, 37, 47, 49-52.) Many cases reported in these papers establish the fact that the difference in uptake of P^{32} by normal tissue and tumor tissue may be used clinically to aid in the differential diagnosis of a malignant melanoma or a more benign condition of the eye.

There are important limitations to the clinical effectiveness of this test. Inflammatory lesions may give false positive readings. Inaccuracies inherent in the counting method may give misleading results, and lesions in the posterior pole may be inaccessible to the counting probe.

All of the reports in the literature to date have concerned themselves primarily with the probe designed for use in the anterior half of the globe. The posterior probe has proved unreliable in actual practice to date. Because of these difficulties, many ophthalmologists are dubious of the value of this test in actual practice. However, the bulk of information available from the analyses to date indicates that when this test is properly performed it is a definite aid in the differential diagnosis of intraocular melanomas. (See table.)

Method

There are several ways which have been recommended for doing this test. A satisfactory one at the present time is that described by Terner, Eisenberg and Leopold.[47] It consists of using approximately 500 microcuries of radioactive phosphorus by intravenous injections. Dunphy employs 750 microcuries of P^{32} intravenously as buffered sodium phosphate. This is the highest dose given to an adult of average size. For small, frail adults 500 microcuries or less may be used. Children under 12 should receive much less. With this dosage the amount of total body irradiation is above that customarily permitted to laboratory workers under daily exposure, but since almost 20 per cent of the P^{32} is excreted in the first twenty-four hours and since its half-life is only fourteen days, it is considered safe.

Readings should be taken with a straight Anton probe counter over the control eye and over the suspected lesion in the affected eye one hour, twenty-four hours, and forty-eight hours after the administration of the phosphorus isotope. Average readings can be taken in each of the four quadrants of the limbus of the normal eye, or control readings may be made at a site on the normal eye which corresponds to the site of the suspected lesion in the fellow eye. Readings in the region of the external rectus of normal eyes may give high counts; this area should be avoided if

RESULTS OF P32 TEST IN ANTERIOR INTRAOCULAR LESIONS ACCESSIBLE TO THE COUNTING PROBE.

(Exclusive of Retinoblastomas and Posterior Inaccessible Lesions)

INVESTIGATOR	NO. OF EYES	POSITIVE	NEGATIVE	FALSE POS.	FALSE NEG.	PATHOLOGY OF FALSE RESULTS
Donn & McTigue (1957)	35	16	17	1	1	(−)Malignant Melanoma-Iris *(small)* (+)Recent Retinopexy.
Dunphy, Dowling, Scott (1957)	31	18	11	1	1	(−)Malignant Melanoma - Iris *(small)* (+)Disciform degeneration.
Palin, Tudway (1955)	13	8	3	1	1	(−)Malignant Melanoma - Iris. *(small)* (+) Hemorrhage.
Thomas, Krohmer, Storaasli (1952)	7	4	3	0	0	
Thomas, Krohmer, Storaasli, Friedhill (1954)	29	11	17	1	0	(+) Uveitis
Owens, King, Weils (1956)	15	11	2	2	0	(+)Recent Retinopexy. (+)Coats's Disease.
Terner, Leopold, Eisenberg (1956)	203	22	175	4	2	(+)Scleritis. (+)Recent Retinopexy. (+) Vitreous Hemorrhage — *(not enucleated)* (+)Absolute glaucoma & uveitis *(not enucleated)* (−)Iris Melanoma *(small)* (−)Diktyoma.
Kennedy, Glasser, Kazdan (1954)	21	9	11	1	0	(+)Intraocular Hemorrhage *(1 hr. reading only.)*
TOTAL →	354	99	239	11	5	

PER CENT ERROR = 4.5% FOR ANTERIOR ACCESSIBLE LESIONS.

FIGURE 1.

possible. In one-eyed persons a quadrant of the eye remote from the site of the lesion is used as a control area. Every effort should be made to hold the probe firmly in apposition to the globe and not tilted. Slight changes in the position of the probe with repeat counts will give different readings. It is therefore important that many repeat counts be taken over each area. It is best to have as little moving about in the room as possible so as not to divert the patient's attention.

Each count should take one minute. The average reading of many one-minute readings at each spot should be employed. This method will decrease some of the errors which occur by virtue of random disintegration of the isotope and the variation due to small changes in position of the probe. In order to consider a reading positive, the value over the suspected lesion must be at least 1.3 times greater than the average reading over the tissue in the control eye. The 24-hour reading should be greater than the one-hour reading. The one-hour reading alone is unreliable, as vascular lesions and inflammatory lesions may give high counts at this time.

There is reason to believe that more accurate differential counts will be obtained in twenty-four hours or more after the injection of P^{32}. During the first fifteen minutes after injection, the phos-

phorus is largely free in the blood stream.[21] During the remainder of the first hour it is diffusing into the tissues. Counts taken at this time only indicate the vascularity of the tissue rather than the rate of cellular growth. There is evidence that after phosphorus diffuses into the tissues, it is found to combine with carbohydrates, fats and proteins. In general, in most tissue studies the combination of radioactive phosphorus is evident first in the carbohydrate, second in the fat portion, and third in the protein.[24] The phosphorus in carbohydrates reaches a state of equilibrium approximately in one hour, whereas the phosphorus and nucleoprotein may take up to forty-eight hours. Malignant tumors contain more nuclei and, hence, more nucleoprotein. It is probably the phosphorus in nucleoproteins that we wish to count; therefore, more rewarding results will be obtained if the count is made at longer intervals after injection. The 24-hour reading is the more important reading. If the results are equivocal, a 48-hour reading can sometimes be helpful. Inflammatory lesions tend to lose their radioactive phosphorus more rapidly than malignant lesions, thus the differential count should be proportionately greater with the passage of time if the lesion is malignant. A dose of 500 microcuries of P^{32} is well within acceptable limits for harmful radioactive effects in the case of adults suffering from probable malignant disease. It is, in fact, of the same order as that received in most x-ray diagnostic procedures. This dose is probably too large to be considered safe for children. The counts are all made following local instillation of 1 per cent Pontocaine Hydrochloride or ophthaine solution. Low counts in normal eyes are very misleading and better results are obtained when high counts are recorded.

The test is of definite value for lesions of the anterior segment involving that portion of the globe which is accessible to the probe.

It is the feeling of this group that posterior lesions may also be tested even though the lesions apparently may not be accessible. A negative result would be interpreted as having no significance. Positive results should provide additional evidence in favor of a malignant melanoma. Release of an extraocular muscle and rotation of the globe will afford direct application of the probe to the posterior segment over the suspected lesion. This area can be compared to remote ones and provide useful information for posterior lesions.

The test has no value in its present form for the differential diagnosis of retinoblastomas. In dealing with blind eyes in which the fundus cannot be visualized the test may also be employed, with the understanding that a negative result means nothing. It should not give a false sense of security, for a tumor may be present which is inaccessible to the probe. A positive result may be helpful.

The P^{32} test gives less accurate results in patients with one eye, in patients with bilateral ocular disease, in patients who have had recent ocular operations and in patients with pathologic changes in the posterior segment.

9. *Are there any hazards to using this test?*

DR. LEOPOLD: The factors which govern the distribution of administered radioactive phosphate have been studied by a number of investigators. General differential uptake of phosphorus by cells is dependent upon at least three factors:

1. The total amount of phosphorus in exchangeable form in the tissue
2. The rate of turnover of phosphorus by the tissue and
3. The rate at which the new tissue is formed

The first point is illustrated by bone. Osseous tissue has a high content of inorganic phosphorus; as a result it eventually takes up a large amount of the radioactive isotope. The effect of the rate of turnover is evidenced from a comparison of the tissues of the liver and brain. Both organs have approximately the same phosphorus content, but the uptake of radioactive phosphorus is much greater in the liver than in the brain because of the higher rate of turnover by the former tissue. The last point is illustrated by the high rate of uptake in the neoplastic tissue as compared with the same type of tissue in a normal state of growth. The tissues which immediately obtain the highest concentration of radioactive phosphorus are the bone marrow, lymph nodes, spleen and liver.

The dosage of 500 microcuries is thought to be below that which will produce depression of the bone marrow function and of lymphoid tissue function. The amount of irradiation produced by this dose is comparable to that required for several roentgen diagnostic procedures, for example, gastrointestinal x-ray series. The total body radiation from this dose is roughly seven times greater than the maximal permissible dose established by the National Committee on Radiation Damage. The amount required for gastrointestinal x-ray series is also more than the maximum permissible dose. This dosage is justified by the National Committee on Radiation Damage, on the ground that the possible diagnostic benefit to the patient outweighs the risk of irradiation. In the case of pregnant women, however, this dose of P^{32} is contraindicated, as embryonic tissue is much more sensitive to radiation than normal adult tissue.

The outstanding effects of overdosage are those that result from depression of bone marrow function with attending leukopenia, thrombocytopenia and anemia. In no case investigated with this dosage in any of the reported series have any of these changes been encountered. Because of the susceptibility of embryonic tissue, the greater possible susceptibility of infants and young children, and the fact that retinoblastomas fail to show consistently positive values with P^{32}, it would seem that for the time being, in pregnant women and young children, this test should be avoided except under special conditions.

Pharmacologic actions of radioactive phosphorus are those which result from ionizing radiations. In all other respects the element behaves in exactly the same manner as a stable isotope. The only radiations released by P^{32} are beta particles; therefore, the effects of radioactive phosphorus in the body are largely confined to the tissues which take up the element. Inasmuch as the immediate uptake of phosphorus is highest in those tissues which normally show the greater sensitivity to ionizing radiation, the effects of radioactive phosphorus are very similar to those of total body radiation.

10. *Are there any other radioactive isotopes that might be employed diagnostically?*

DR. LEOPOLD: A number of radioactive isotopes have been investigated in an effort to find out which one of these would be taken up more readily by tumor tissue. Various radioactive isotopes such as gallium (Ga^{72}), copper (Cu^{64}), iodine (I^{131}) have been tried. When a tumor is superficially approachable, as it is in the ocular tissues, a beta emitting radioactive substance is far more useful than a gamma emitter. Short range beta emissions of P^{32} would be superior, therefore, to the gamma emissions of I^{131}. The gamma rays have a penetrating power of several centimeters, and the radiation from distant sites would mask the local response from a small tumor

site. The high general response from all neighboring tissues would mask the small tumor. This observation can account for the negative findings of Trever-Roper, Neuton and Nicholson, who used I^{131}.

Other radioactive isotopes which have been employed in diagnostic tests and as tools for research in the fundamental biology of disease include radioactive sulfur (S^{35}), radioactive potassium (K^{41}), rhubidium (Rb^{86}) and radioactive iron (Fe^{59}).

O'Rourke, Patton and Bradley[34] have studied uptake of zinc (Zn^{65}), as well as other radioactive isotopes, by malignant melanoma experimentally induced in animals. Although these other radioactive isotopes are available, they have not approached the stage of practicability that has been reached by P^{32}.

11. *Dr. Forrest, what place does biopsy have in the diagnosis of melanoma of the uveal tract?*

DR. FORREST: Biopsy of the uveal tract for diagnosis of tumor has many limitations and very few indications. In the case of iris tumors in which complete excision is not feasible because of the extensiveness of the tumor, this clinical behavior has indicated the diagnosis and surgical meddling would be unwise.

Biopsy of the ciliary body or choroid is almost never indicated; the clinical methods discussed earlier generally give enough information to determine whether therapy is to be aggressive or expectant even without the final diagnosis.

Our attitude toward intraocular biopsy is determined mainly by three features about which Sanders[43] has written.

A. The lack of real value in the usual case of suspected tumor

B. The possibility of disseminating the tumor from manipulation during biopsy

C. The possibility of doing damage to the eye

A. Large tumors generally do not present diagnostic problems. Occasionally ocular function has been compromised by a large tumor so that enucleation may be indicated even without final evidence of tumor. If the tumor is small and flat, the technique of obtaining enough material for reliable interpretation may be dangerous to the eye. False negative reports would be common so that, in those instances in which the clinical diagnosis is not clear, exposing the eye to the further insults of the biopsy attempt may add little information.

When the amount of pathologic material received is small, it can only be studied cytologically. In contrast with the vast experience with the Papanicolaou method in study of the uterine cervix, a method which is used extensively with massive accumulation of data, so rarely is the method used in the eye that the interpretation is open to considerable question. Further, as Dr. Zimmerman will show, small tumors are likely to be of a more benign type in which the cytologic diagnosis of malignant growth would be especially difficult. Enough material to be adequate for histopathologic preparation would be difficult to obtain from small intraocular tumors.

B. In reference to the possibility of spread of tumor during biopsy, although instances of survival are recorded when enucleation followed immediately upon positive biopsy, episcleral nodules of tumor have developed when enucleation was delayed for weeks or even days. From this point of view also, biopsy is not innocuous.

C. Damage to the eye can occur because of biopsy procedures, especially when the tumor is small. Whether the

approach to the tumor is by direct sclerotomy or across the vitreous, whether the tissue is acquired by aspiration, curettage, trephination or prolapse of tumor into the sclerotomy wound, such complications as intraocular hemorrhage, loss of vitreous or retinal detachment can occur.

Rarely, tissue may be removed inadvertently in aspirating ciliary body cysts or in draining fluid from supposed idiopathic detachment.

In summary, an iris tumor should be excised completely if it is localized and does not involve the angle. If the iris tumor is massive, extremely diffuse or invasive, the diagnosis is clear and biopsy is not indicated. The dangers and unreliability of biopsy of ciliary body and choroidal tumors minimize its value.

12. *How about subretinal fluid aspiration in retinal detachment when tumor is suspected? Would you tell us whether this has been used to any extent and whether you think it justified?*

DR. FORREST: The aspiration and study of subretinal fluid in retinal detachment with suspected tumor, in contrast with biopsy, is a relatively innocuous procedure, although not always any more enlightening. There are several reported instances in which tumor cells were identified when choroidal tumor was present, and others in which, without tumor, these cells were absent. The method requires microcentrifugation and examination by cytologic techniques. None of these reports make clear that the eyes were diagnostic problems, and as Reese[39] has stated, in the early stages which are generally a greater diagnostic problem, cells are not likely to be found in the subretinal fluid. In these cases the tumor remains within the confines of the lamina vitrea and tumor cells would not be expected in the subretinal space. It would seem, also, that normally pigmented cells, pigment epithelium or nonpigmented tumor cells could give false results. Christensen[8] studied several eyes and concluded that the differentiation of tumor cells from pigmented histiocytes was uncertain and that if a tumor-bearing eye were left in situ for as little as thirteen days, the tumor could extend outside the eye.

Cibis[9] has reported positive results in tests for melanin in subretinal fluid in the presence of melanoma, and negative tests on intraocular fluid in the absence of tumor. Blodi[5] has seen an instance of a false negative test result.

Parenthetically, tumor cells have been described in the aqueous, but this test is seldom used.

The reliability of the results of study of subretinal fluid is too low to justify its use except in unusual instances.

13. *The use of ultrasonic waves has been suggested as a means of diagnosing intraocular tumors. Dr. Leopold, can you tell us whether this has been sufficiently developed to be practical?*

DR. LEOPOLD: Localization of tumors by ultrasonic reflection has been performed on tissues of the body other than the eye by a number of investigators.[2,20,23,32,41]

Ultrasonic waves are sound waves which have a frequency above the hearing range of the human ear. This includes all vibrational waves above approximately 20,000 cycles per second. Ultrasonic waves of higher frequency have many propagation features similar to the characteristics of light waves. They tend to travel in a straight line and may be focused by using a concave prism. When an ultrasonic beam passes into a medium with characteristics different from the one in which it came, some of it is refracted and some reflected. The amount depends on the difference in the density and the elasticity of the

two mediums and the angle at which the beam strikes the new surface.

An ultrasonic "reflectoscope" is used for the detection of such things as gallstones. It has been demonstrated that large signals would be returned from almost any metal, wood, glass or plastic foreign body imbedded in soft tissues. Reid and Wilde[41] have reported that malignant breast tumors showed the largest echoes on the reflectoscope, normal tissue next, and benign tumors the smallest reflections. For this reason, Mundt and Hughes[32] investigated the use of the reflectoscope as an aid in helping to differentiate a serous retinal detachment from one that is the result of a tumor. They concluded that this is a tool with which one is able to detect the distances of various reflecting surfaces within the eye. At the present time, this can be done with an accuracy not greater than 1 or possibly 2 mm. With this instrument, simple serous detachments can possibly be differentiated from those due to tumors, but it has not been determined whether tumors can be differentiated from such things as hemorrhages and scars.

Although the ultrasonics have some potential value for localization of tumors intraocularly, the technique must be developed and improved before it reaches a useful position. Definite knowledge has to be gained concerning the necessary megacycles of ultrasonic energy to detect orbital and ocular abnormalities and the tolerance of ocular tissues for this required energy; then, a functioning instrument for routine ocular purposes must be created. Such studies are underway by a number of investigators, such as Mundt and Hughes, and Baum and Greenwood.

14. *Dr. Forrest, tell us how you think slit lamp examination and gonioscopy can be employed to advantage in the diagnosis of iris melanomas.*

DR. FORREST: Lesions of the iris in early stages generally produce no functional defects, there may be no visual changes, and radioactive phosphorus uptake may be normal; their diagnosis depends upon the interpretation of their appearance. This may not always be conclusive, but in addition to gross examination and serial photography, the magnification of the slit lamp microscope and the view afforded by the gonioscope are indispensable.

Slit Lamp

1. The biomicroscope, clinical drawings and photographs reveal the most important single factor in the diagnosis of malignancy in an iris tumor—namely, growth. It is possible with this magnification to appreciate extremely small increases in size in relation to minute anatomic landmarks. Malignant melanoma of the iris is generally more extensive than the benign melanoma or freckle, and the biomicroscope is helpful in demonstrating, when it is present, the elevation of the surface of the iris. Benign lesions generally do not protrude above the iris surface. Their loose texture in contrast to the fleshy appearance of the malignant lesion also can be best identified with magnification.

2. In the case of heterochromia, the slit lamp is helpful in evaluating whether the eye with less pigment is the bed of inflammation or whether the darker eye is the site of increased pigmentation. In the latter instance, when the pigmentation is due to tumor, the biomicroscope shows diffuse increased pigmentation, a thickened iris, the loss of normal iris markings, and the filling in of crypts.

3. The biomicroscope aids in identifying the implantation masses of malignant tumor when it can be seen that the bulk of the seed lies upon the surface of the iris.

4. Evidence of local extension of malignant tumor in the iris can best be seen with magnification. Benign lesions generally have regular borders without the ramifications and irregular extension into surrounding tissue which characterize some malignant tumors.

5. The biomicroscope reveals the prominent vascularization in and about malignant tumors. The presence of many large blood vessels which may extend into the stroma of a tumor suggest that it is malignant and sometimes that it is undergoing active growth.

Gonioscopy

However important gonioscopic examination may be in the management of glaucoma and other ocular disease, it offers invaluable service in the diagnosis of tumors of the iris.

1. Evidence of growth of peripheral tumor, as indicated by change in elevation or its borders, is best measured by gonioscopic examination.

2. The extensiveness of the tumor as it determines the feasibility and extent of an operation can only be known after study of the peripheral iris.

3. Cancer cells growing in the angle as implantation foci or as extension may be identified by gonioscopic examination. These can sometimes be differentiated from pigment by their denser agglomerated appearance, in contrast to the diffuse powdery nature of extracellular pigment. The seeds of tumor are most conspicuous in the angle below.

Careful study of the patient with glaucoma in an eye with increased pigmentation may show that the ring of pigment is most intense in the trabecular area, suggesting that the glaucoma is of pigmentary origin. When the glaucoma is related to tumor, the pigmentation involves all of the angle in an irregular manner. This can be studied with the gonioscope.

Dr. Leopold has already mentioned the use of the biomicroscope and gonioscopes in studying transillumination.

15. *Dr. Reese, what are the lesions of the iris and ciliary body which require differentiation from malignant melanoma?*

Dr. Reese: The differentiation usually rests between the following lesions:

1. Intraepithelial cyst
2. Leiomyoma or leiomyosarcoma
3. Granuloma
4. Encysted foreign body
5. Epithelioma of the ciliary body
6. Implantation or cyst of the pigment epithelium
7. Localized broad anterior synechias
8. Staphyloma or localized ectasia
9. Metastatic tumor
10. Dictyoma
11. Lymphosarcoma
12. Melanosis oculi (congenital heterochromia of the iris)
13. Partial melanosis oculi (congenital heterochromia of the iris)

16. *Dr. Zimmerman, what are some of the other lesions found in eyes enucleated for melanoma of iris and ciliary body?*

Dr. Zimmerman: The wide variety of lesions which may be confused with malignant melanoma of the iris and ciliary body can be placed in two categories. One includes such discrete lesions as staphylomas, cyst of the anterior and posterior chambers, granulomas, foreign bodies, nevi, hemangiomas, and leiomyomas. The other includes more diffuse processes leading to alteration of the pigmentary characteristics of the iris, heterochromia iridis or heterochromia iridum. Siderosis bulbi from a ferrous foreign body, hemosiderosis sec-

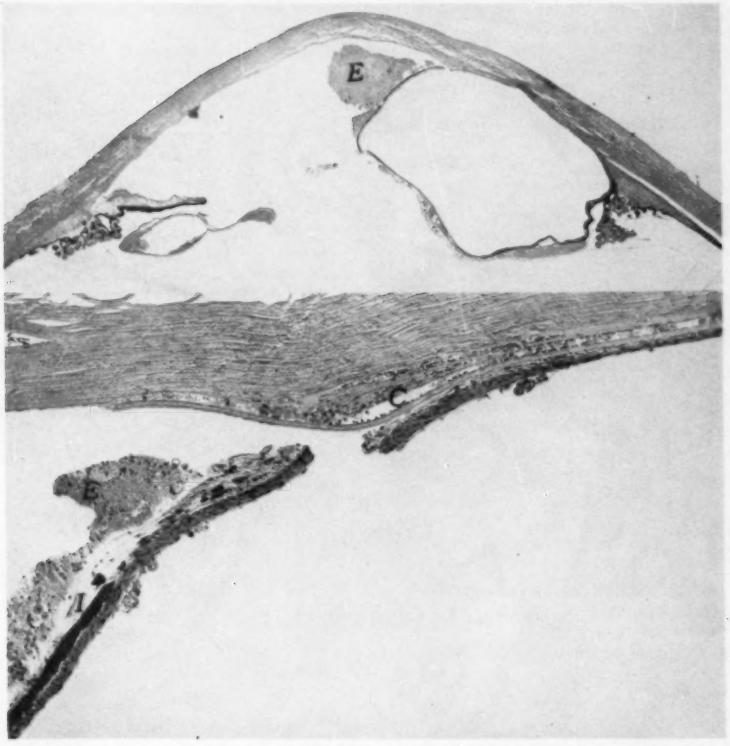

FIG. 16-1—AFIP Acc. 238572. (Top) Epithelial cyst of anterior chamber, the result of downgrowth of surface epithelium following extracapsular cataract extraction. (H & E, × 7) (Bottom) The cyst is lined by stratified squamous epithelium and covered by the atrophic iris (I) except where it is in contact with the cornea (C). A mass of exudate (E) clings to the mesial surface of the cyst. (H & E, × 90)

ondary to repeated intraocular hemorrhage, necrosis of the iris stroma permitting the pigment epithelium to be visualized more directly, and benign melanosis of the iris are examples of some of the conditions I have seen which give the false impression of malignant melanoma. Illustrative cases will now be given.

AFIP Acc. 238572. A 74-year-old white man underwent an extracapsular cataract extraction in 1944 and a needling operation in 1947. A lesion on the iris was first noted in October 1948. It was described as a yellow mass growing backward at the nasal pillar of the operative coloboma. It was suspected to be a "sarcoma of the ciliary body," and the eye was enucleated in February 1949. Pathologic study revealed it to be a cyst formed by epithelial downgrowth into the anterior chamber (fig. 16-1).

Staphylomas, as I have said earlier, may be misinterpreted as melanomas. In the following case a staphyloma of the cornea was believed to be a melanoma of the iris:

AFIP Acc. 613664. A 63-year-old white man was first seen by an ophthalmologist in

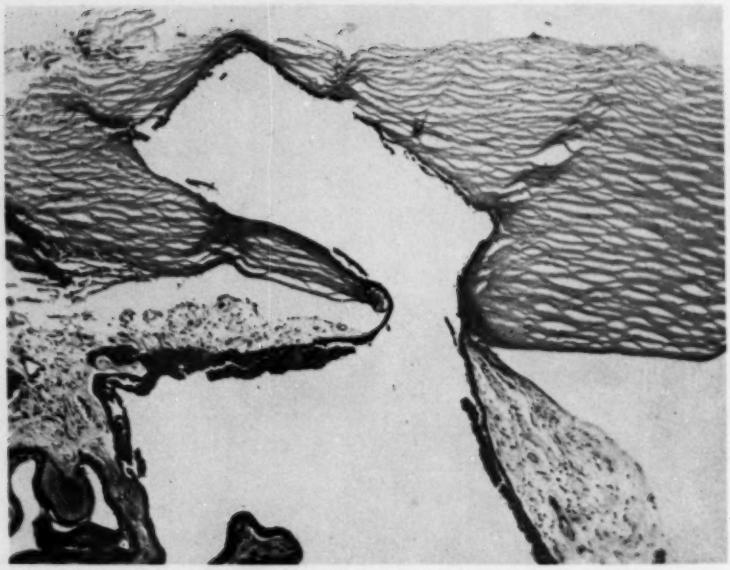

FIG. 16-2—AFIP Acc. 613664. Atrophic iris epithelium lines a cystoid scar of the peripheral cornea. (H & E, × 56)

FIG. 16-3—AFIP Acc. 765506. There is widespread malformation of the iris, ciliary body, and retina on the nasal side. A cyst lined by pigment epithelium projects into the posterior chamber, where clinically it could be visualized through the pupil. (H & E, × 11)

December 1952, at which time no significant pathologic process was found. Nine months later he returned to the same ophthalmologist because of bilateral allergic conjunctivitis. In the course of this examination a discrete black lesion, 1 mm. in diameter, was noted in the limbus. A diagnosis of malignant melanoma of the iris was made. The patient was examined in consultation at a well-known university department of ophthalmology, where the clinical diagnosis was confirmed. Enucleation was performed a few days later. Histologic study revealed a focal defect in the peripheral cornea (fig. 16-2) and iris incarcerated in the wound. Neither an intraocular neoplasm nor other ocular lesion was present.

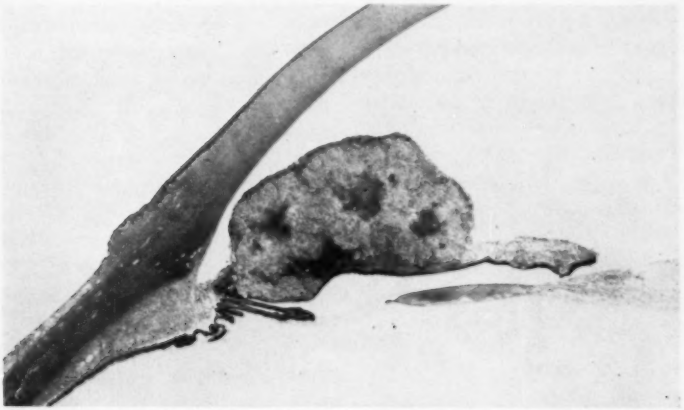

FIG. 16-4—AFIP Acc. 119782. Discrete granuloma of iris, cause undetermined. (H & E, × 16)

AFIP Acc. 765506. A 22-year-old white woman first noted loss of the temporal field of vision in her left eye two days before admission. Examination revealed a small dark mass at the nasal margin of the pupil which caused slight subluxation of the lens. The nasal retina was detached, and there was a hole in the upper nasal quadrant. Vision was reduced to light perception, but the tension was normal. The clinical diagnoses were (1) detachment of retina with hole in upper nasal quadrant, and (2) neoplasm of ciliary body. The eye was enucleated ten days after the onset of symptoms. Examination in the laboratory revealed large flat thickened ciliary processes extending onto the posterior lens surface for a distance of 2 mm. on the nasal side. Microscopically widespread malformation of the intraocular structures on the nasal side was observed. The iris root arose far back on the ciliary body. Huge, bizarre ciliary processes, covered anteriorly by pigment epithelium and posteriorly by nonpigmented ciliary epithelium and by sensory retina, projected into the posterior chamber behind the lens equator (fig. 16-3). Some sections also revealed cysts of the pigment epithelium in the posterior chamber behind the nasal side of the iris. A small tubelike mass of glial tissue was present behind the lens in the region of Cloquet's canal. Flat detachment, widespread microcystoid degeneration, and loss of rods and cones were observed throughout the entire retina on the nasal side; the temporal retina remained well preserved.

AFIP Acc. 119782. The left eye of a 6-year-old white boy was enucleated because of two discrete masses on the anterior surface of the iris which had been diagnosed "sarcoma." Histologically the lesions proved to be granulomatous and consistent with tuberculosis (fig. 16-4), although acid-fast organisms were not found.

AFIP Acc. 786483. An 83-year-old white man complained of failing vision of the left eye of ten years' duration when he was first seen in 1951. At that time he had a subluxated

lens with a mature cataract and secondary glaucoma. He refused cataract extraction. When seen again five years later, he had absolute glaucoma and the anterior chamber was very shallow. An area of deep-brown pigmentation involving the peripheral half of the iris in the lower temporal quadrant was believed to represent a malignant melanoma of the ciliary body. Enucleation was performed. On transillumination of the eye in the laboratory the discolored area was seen to be extremely atrophic iris which was much more translucent than the remainder of the iris. This interpretation was confirmed by histologic study, which revealed complete degeneration of the iris stroma and the pigment epithelium plastered against the cornea (fig. 16-5).

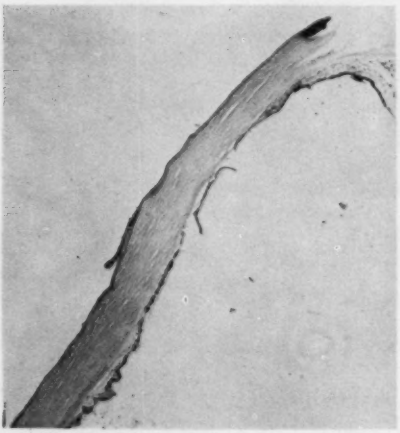

FIG. 16-5—AFIP Acc. 786483. The extremely degenerated iris is firmly adherent to the cornea. Over a large area the pigment epithelium is in direct contact with the cornea. (H & E, × 15)

17. *Dr. Zimmerman, you have told us about the various lesions found in eyes removed because of the suspicion of tumor. What can you tell us about clinically unsuspected intraocular melanomas?*

DR. ZIMMERMAN: I have been surprised both in my own experience and in that of others to find how often melanomas are discovered only after the enucleated eye is opened in the laboratory. In a study of 228 cases of malignant melanoma of the choroid on file in the pathology laboratories of the Illinois Eye and Ear Infirmary and the Research and Educational Hospitals of the University of Illinois, Kirk and Petty[26] found that 24 (10.4 per cent) were not suspected clinically. Nearly the same incidence was found in a much larger series of intraocular melanomas reviewed at the Armed Forces Institute of Pathology by Makley and Teed.[30] These investigators found that 212 of 1,000 eyes with malignant melanomas had opaque media which prevented visualization of the ocular fundus. In 99 a tumor was suspected, but in the remaining 113 (11.3 per cent), there was no suspicion of tumor until the enucleated eye was examined in the laboratory. In Reese's series from the Institute of Ophthalmology, Presbyterian Hospital, New York, the incidence of unrecognized malignant melanoma was lower.[38] He found 18 cases (6.7 per cent) in which the clinical diagnosis was glaucoma, retinal detachment, non-neoplastic macular lesion, or phthisis.

Summing up these three series we may say that malignant melanoma was not suspected clinically in about 10 per cent of almost 1,500 cases in which the presence of the tumor was proved pathologically (table I).

We might wonder why the incidence of unrecognized tumors should be so high. Almost all were found in eyes with opaque media, eyes which could not be examined satisfactorily. Makley and Teed,[30] by the way, found that in almost 4 per cent of 1,000 adult eyes with opaque media and blind for six months or more, malignant melanomas were discovered upon pathologic examination.

TABLE I

INVESTIGATORS	CASES OF PROVED INTRAOCULAR MALIGNANT MELANOMA	UNSUSPECTED CASES	
		NO.	%
Reese, 1951	271	18	6.7
Kirk and Petty, 1956	228	24	10.4
Makley and Teed, 1957	1000	113	11.3
Totals	1499	155	10.3

The next question that arises is "Why were the media opaque?" There are two main reasons. First, the eye may have been previously damaged by trauma or

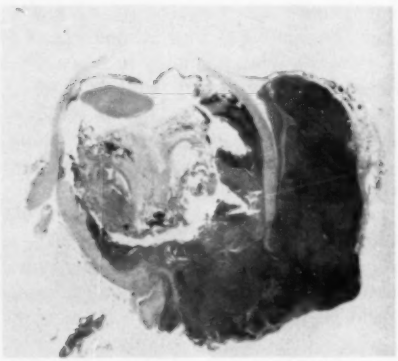

FIG. 17-1—AFIP Acc. 24068. A metallic foreign body is contained within the organized vitreous mass of this phthisical eye, which was injured twenty-two years before enucleation. The melanoma of the posterior choroid has broken out of the globe to form a large orbital mass. (H & E, × 4)

disease. Malignant melanoma can develop in any eye, be it normal, phthisical, or glaucomatous. An example is AFIP Acc. 24068 (fig. 17-1). This shrunken eye was injured twenty-two years before enucleation. A retained metallic foreign body was buried in the organized vitreous and the lens was cataractous. A malignant melanoma arose in the posterior choroid of this phthisical eye, but it was not recognized until extraocular extension produced an orbital mass which caused proptosis. On the other hand, I believe that more often the hidden melanoma itself is responsible for such changes as retinal detachment, vitreous degeneration, opacities of the lens and corneal edema which cause the media to become opaque. Probably the most important factor is necrosis of the tumor, for necrotic melanoma tissue is extremely irritating and it will provoke an inflammatory reaction which can be mistaken for infectious endophthalmitis or panophthalmitis. Necrotic melanomas are also more likely to cause severe intraocular hemorrhage, and they are much more often associated with secondary glaucoma. For all of these reasons, necrotic melanomas are frequently complicated by opacified vitreous, mature cataract, corneal edema or keratitis. Makley and Teed found that in almost one-fourth of the 212 melanoma cases in which the fundus could not be visualized because of opaque media, the tumor was too necrotic to be classified according to cell type.

There is one important exception to the generalization that when melanomas are not recognized, it is because the media are opaque. The exception is the diffuse type of malignant melanoma of the iris. This tumor may arise so insidiously that the heterochromia which it produces escapes detection. The patient is treated for glaucoma, sometimes with several operations, before it is realized

that a tumor infiltrating the anterior chamber angle is responsible for the impaired aqueous outflow.[42]

Finally, I would like to mention one additional cause for confusion in the clinical diagnosis of intraocular melanoma. Patients with a history of carcinoma, especially women who have had a radical mastectomy for breast cancer, may also have an intraocular melanoma. A sizable group of such cases can be found in the Registry of Ophthalmic Pathology. Enucleation may be withheld in the belief that the tumor is metastatic carcinoma. Although experienced ophthalmologists feel that metastatic carcinoma can usually be differentiated from malignant melanoma, I personally know of a number of cases in which these two tumors could not be correctly differentiated until histopathologic study had been completed. The error can be in either direction: melanomas may simulate metastatic carcinoma when they are flat, diffuse growths, and metastatic carcinoma may produce a large collar-button-like mass characteristic of the choroidal melanoma, as we have just shown.

18. *Dr. Leopold, in view of what Dr. Zimmerman has told us, do you think that all blind eyes should be removed as a general precaution?*

DR. LEOPOLD: There can be no doubt that some blind eyes harbor unsuspected malignant melanomas, but one cannot say that all blind eyes should be removed as a general precaution. There are several reasons for this: In a recent large series (Ashton) of 862 eyes which were removed with a clinical diagnosis of malignant melanoma, 102 of them (about 12 per cent) proved to be nonmalignant. Many times, the ophthalmologist who removes all blind eyes will be sacrificing an eye that does not harbor a tumor. In many of these blind eyes the cause of blindness has been well documented by careful study over years of observation, and in many the fundi have been examined carefully and intraocular malignancy can be eliminated fairly well as a possible diagnosis. If the ophthalmologist can be reasonably sure of the cause of the blindness and has or had recent access to a fairly satisfactory fundus examination, and if there are no other reasons for suspecting an intraocular malignant lesion, it does not seem reasonable to advise enucleation, particularly when the patient is comfortable. Excellent as many of the replacements are for enucleated eyes, they may not improve the cosmetic situation and their care does offer an additional burden to the patient. On the other hand, painful blind eyes should be removed, particularly when the fundus cannot be seen. If one is in doubt, he may employ a radioactive phosphorus test. If this test is negative, this should give him no sense of security that a tumor is not present, for the probe may miss a small tumor and the probe may not reach the tumor site. A positive result, however, would suggest the possibility of an intraocular malignant lesion and give further reason for enucleation of a blind eye. Enucleation should be performed only in those eyes in which the cause of blindness cannot be established, the eye cannot be adequately examined because of opaque media, or there is some other reason for enucleation, such as uncontrollable pain.

Unfortunately, an eye blinded by non-neoplastic cause is not protected from the development of a malignant melanoma. According to statistics of Makley and Teed gathered at the Armed Forces Institute of Pathology, of 969 patients 30 years old or older with eyes blind for more than six months' duration that came to enucleation, 37 or almost 4 per cent harbored malignant melanoma. In a series of 300 eyes enucleated between April 1954 and December 1956 at Wills Hospital, Knisely found that 5 per cent of all of the blind eyes

contained malignant melanomas and 3 per cent of all blind and painful eyes contained malignant melanomas. Pain is not a usual accompaniment of intraocular melanoma nor does it exclude the presence of an intraocular tumor.

DR. DUNPHY: Let us now pass on to the management of these cases, assuming that the correct diagnosis has been made. Let us assume that we are dealing with a case of malignant melanoma. Shall we enucleate immediately or shall we consider some other method of attack?

Our decision will depend on many factors relating to the patient's age, physical condition, size of the tumor, and condition of the other eye. For a one-eyed individual a decision to enucleate cannot be made lightly. To help us decide we must have some appreciation of the chances of metastasis. We know that some tumors never metastasize.

19. *Dr. Zimmerman, on the basis of your pathologic experience, tell us on what real evidence enucleation is advised for malignant melanoma of the choroid and ciliary body.*

DR. ZIMMERMAN: The only "real evidence" to a pathologist is that obtained from microscopic examination of the tissue in question. But biopsy, as Dr. Forrest has already told us, is not feasible in the case of tumors of the choroid and ciliary body. Since most of the factors that are known to influence prognosis are determined by histopathologic study, these are of no value to the clinician before the eye is enucleated. There is, however, at least one important prognostic factor which can be used clinically; i.e., size of the tumor.

In a study based on 210 melanomas of the choroid and ciliary body, it was shown that the 105 patients with the largest tumors had a significantly poorer prognosis than the 105 patients with small tumors.[18] The reason for this, I believe, is that tumor size gives some index of cell type. Small tumors are usually made up of spindle cells, while large tumors contain a significantly higher percentage of epithelioid cells. Because the prognosis for patients with tumors measuring less than five disc diameters is generally so favorable, I feel quite strongly that an ophthalmologist should be extremely certain of his diagnosis before recommending enucleation for a small lesion. It is fortunate that these small tumors carry the best prognoses, for it is the small lesion that gives the greatest difficulty in clinical diagnosis; also, eyes containing such small tumors are more likely to have useful vision. I would have no hesitation in recommending that a patient with a small questionable lesion be placed under observation and that serial photography, visual field studies, and any other diagnostic procedure considered helpful be employed until there is unquestionable evidence of progression and the clinical diagnosis of malignant melanoma is indisputable.

In a thought-provoking paper dealing with prognosis of melanomas of the choroid, Westerveld-Brandon and Zeeman[53] raised the question as to what we are accomplishing by enucleation, especially in patients over 60 years of age. They found that only three of 23 such patients reached an age compatible with their expectation of life; nineteen died of metastasis and one of intercurrent disease at an earlier age. Furthermore, these authors pointed out that although their younger patients were faced with a much less gloomy outlook, they nevertheless might not live to their normal life expectancy. Therefore, Westerveld-Brandon and Zeeman have apparently abandoned the conventional practice of recommending early enucleation in the case of patients aged

60 or more and they are not even convinced that it is justifiable for younger patients who still retain useful vision.

Obviously, what we need in order to determine the influence of enucleation on life expectancy is a follow-up study of a group of patients with intraocular melanomas who are not treated by any method along with another group treated by the conventional early enucleation.

20. *Dr. Reese, from a clinical point of view what is your opinion?*

DR. REESE: I agree with Dr. Zimmerman so much that I would like to emphasize something he has said by quoting him:

> Small tumors are usually pure spindle-cell types, while large tumors contain a significantly higher percentage of epithelioid cells. Because the prognosis for patients with tumors measuring less than five disc diameters is generally so favorable, I feel quite strongly that an ophthalmologist should be extremely certain of his diagnosis before recommending enucleation for a small lesion.

To illustrate how slowly some malignant melanomas of the choroid may grow I cite the following cases:

Case 1

Twenty-two years before an eye was enucleated there was a solid detachment of the retina with poor vision. The clinical diagnosis was malignant melanoma of the choroid. The tumor was estimated to be 2 to 3 disc diameters in diameter and elevated 12 diopters. The patient refused enucleation. At yearly observations slow progression was noted. Finally, enucleation was done by Dr. Jesus Rhode of Caracas, Venezuela, because of glaucoma and pain. The patient died three years ago of cardiac disease with no evidence of local recurrence or of distal metastatic lesions six years after enucleation and 22 years after the original diagnosis. The tumor was a spindle-cell malignant melanoma of the choroid which filled the eyeball.

Case 2

The clinical diagnosis of malignant melanoma of the iris and ciliary body was made twenty-seven years before the eye was obtained for sectioning. A cataract extraction, which resulted in normal vision, was done eight years before by Dr. Ivan Koenig,[27] who reported this case. The globe was obtained for sectioning when the patient died of coronary occlusion without evidence of tumor growth or dissemination. The tumor, which had shown no appreciable growth, was a malignant lesion of the epithelioid type.

In the first case a tumor made up of spindle cells grew very slowly and manifested no metastases over a period of twenty-two years. In the second case a tumor of the epithelioid type showed no appreciable growth and no metastases for twenty-seven years.

21. *Dr. Zimmerman, are iris tumors as malignant as choridal ones?*

DR. ZIMMERMAN: No, definitely not! The majority of iris tumors are small spindle cell melanomas which, if completely removed by iridectomy, will not recur or metastasize. When incompletely excised, they will recur, but rarely do they metastasize. This aspect of iris tumors was brought out dramatically by a study of diffuse iris melanomas reported at last year's Academy meeting by Dr. Ben Rones and me.[42] We collected 66 such cases and were successful in obtaining follow-up data on 46. Only 6 patients had died and only 3 of these deaths were from metastatic tumor. Of the 40 living patients, 24 had been followed five to fifteen years after enucleation.

22. *Dr. Zimmerman, do you believe that the diagnosis of a uveal melanoma constitutes a surgical emergency?*

DR. ZIMMERMAN: No, I don't believe that the handling of a case of suspected intraocular melanoma should be considered a surgical emergency. This, as I indicated a few moments ago, is especially true of small tumors of the choroid and ciliary body and of most iris melanomas. Here is a place where haste may waste a useful eye. Though I have no statistics to prove my point, I believe

I have seen more damage resulting from the desire to enucleate early than from delayed operations.

23. *Dr. Reese, do you think that pregnancy can influence the genesis or the course of a malignant melanoma?*

DR. REESE: It is well known that pigmentary changes do occur in the skin of the face, nipple, and abdomen of pregnant women. Furthermore, existent nevi may show darkening during pregnancy. However, these pigment changes cannot be viewed as tantamount to a predisposition to develop melanoma.

There has been some evidence reported that ACTH and cortisone may produce junctional nevi which may go on to malignant melanoma. As the level of ACTH is increased in pregnancy, there is a need for its appraisal in the question posed here.

Furthermore, it has been shown that there is a increase in the excretion of a melanocyte-stimulating hormone (MSH) after the second month of pregnancy and a sudden decrease after delivery of the infant. It has been suggested that the activation of pigmented nevi and melanoma during pregnancy may be related to an increased secretion of MSH. Whether this increased MSH level in patients with malignant melanoma is a primary or secondary factor has not been established.

Therapeutic abortion will not benefit a mother with a uveal melanoma.

Malignant melanoma may be transmitted from the mother to the fetus in utero, but the hazard is extremely remote.

Clinical evidence indicates that pregnancy accelerates the growth of a preexisting melanoma rather than having a bearing on the genesis of the tumor.

24. *Dr. Reese, would you consider coagulating a malignant melanoma of the choroid with diathermy in a patient's only useful eye?*

DR. REESE: I doubt whether diathermy has any place in the therapy of uveal melanoma.

The reported cases are not convincing as to its efficacy on the following grounds:

1. It is frequently not clear that the lesions treated were malignant melanomas.
2. When the lesions were small—1 to 2 disc diameters in size, active growth was not definitely established before treatment was instigated.
3. Enucleation was necessary in four instances because of continued growth, and two of these patients died within one year following the enucleation.
4. The follow-up period is too short. In one it was one year and in another it was six months.
5. Useful vision is not obtained due to the detachment of the retina and other sequelae.

Furthermore, the treatment by perforating diathermy which is necessary to reach the desired depth is a serious hazard on two counts: (1) There is good evidence that tumor growth is accelerated. This is gathered, for the most part, from the cases of uveal melanoma operated on with the erroneous diagnosis of serous detachment. (2) Viable cancer cells may be seeded in the perforations and the surrounding orbital tissues.

Conclusion from two cases cited in which a diathermy operation had been done on eyes with malignant melanoma with the belief that serous detachment was present:

1. On the basis of the usual rate of growth of uveal melanomas, we can say that growth was accelerated.
2. The operations were no doubt responsible for orbital extension.

3. The operations were probably an important factor in the early demise of both patients from metastatic tumor.

Certainly diathermy should be considered only in the case in which the only useful eye is involved. Even here it seems inadvisable.

Following are the circumstances where it might be employed:

1. The lesion is a benign, not a malignant, melanoma for which no treatment is indicated.
2. The lesion is a malignant melanoma but of the spindle-cell type, which may grow extremely slowly, as pointed out by Dr. Zimmerman. The patient may retain useful vision for ten to twenty years without jeopardy to life. Enucleation could be done when useful vision is lost.
3. The lesion is a melanoma with a more malignant type of cell structure. If or when useful vision is lost, enucleation could be done.
4. The condition proves not to be a melanoma but a simulating lesion—such as, inflammation, degeneration, cyst, hemorrhage, metastatic neoplasm, hemangioma. In such instances diathermy would be contraindicated.

On the basis of the present evidence we are unable to recommend diathermy even for one-eyed patients.

25. *Every once in a while one hears a report of successful treatment of a malignant melanoma by irradiation. Dr. Forrest, will you tell us the current feeling on the subject by those with experience in radiotherapy?*

DR. FORREST: Malignant melanoma, in general, is not radiosensitive; hence, the use of radiation is limited to rare instances of tumor in an only useful eye. Radiation of uveal melanomas is not known in this country, where roentgen ray is the more commonly used moiety, but it has been reported in several instances by our British colleagues using the gamma radiation of gold radon seeds. Earlier, these were placed directly in the tumor at operation; but as the tumor became necrotic, the seeds were released into the vitreous and the eye was compromised by further mechanical trauma and by radiation. More recently the radon seeds have been sutured to the sclera. Stallard[45] has reported an instance of a choroidal lesion diagnosed as malignant melanoma clinically which, ten years after radiation, had shrunken to a flat pigmented area one-eighth of its original size, surrounded by atrophic retina and choroid. A few vacuoles at the equator of the lens in one quadrant had not changed in seven years, and there was no visual impairment.

Reports of successes in the world of literature number only a very few, and in none of these, by necessity, is there histologic documentation of the diagnosis of malignant melanoma. Radiation is not to be considered as a method of treatment of uveal malignant melanoma.

26. *And now, Dr. Forrest, how about chemotherapy? We know it is valuable in retinoblastoma; do you know of any chemotherapeutic agent that can favorably influence a malignant melanoma?*

DR. FORREST: In general, with our present knowledge the ophthalmologist will not have occasion to use chemotherapy in the treatment of uveal malignant melanoma. In all the work which is being done on cancer chemotherapy, melanomas represent an extremely small part of the tumor groups, and uveal melanomas are almost unknown, so factual data on this particular tumor is sparse. However, other melanomas have been treated and the results are poor.

Throughout the literature on chemotherapy we find scattered reference to the idea expressed in the following quotation by Farber and his colleagues[17]:

> [The drug (Tepa)] was administered, as is customary in a program of clinical investigation, as part of the total care to patients either with cancer too far advanced to permit beneficial treatment by the techniques of surgery or roentgenray therapy or with forms of cancer such as acute leukemia for which no cure is available.

It is therefore apparent that at this time intraocular melanoma is not even being considered in experimentation on chemotherapy.

Several groups of chemicals have been used in tumor therapy. Of these, in patients with disseminated disease, temporary improvement has been noted with some of the ethylenimines and with nitrogen mustard and corticosteroids. There were no instances of cures, but occasionally either a temporary decrease in the gross size of the metastatic nodule was noted or biopsy showed some central necrosis. In any case, with our present information chemotherapy has nothing to add to traditional methods of treating uveal malignant melanoma limited to one of two eyes. In an only eye, or in tumor residual or recurrent in the orbit, chemotherapy under the guidance of the oncologist may be considered, but this method of treatment remains experimental and at present there is no basis for optimism about the therapeutic results.

27. *One more question, Dr. Forrest. Recently Dr. Meyer-Schwickerath of Germany has used heliocautery to seal retinal tears in detachment cases and has also employed this method to attack choroidal melanomas. Do you think this form of treatment might have a future?*

DR. FORREST: The use of heliocautery is such a new and revolutionary idea and the apparatus so complex that only Dr. Meyer-Schwickerath has had any experience with this technique of treating melanomas of the choroid. I therefore quote from the works of Dr. Meyer-Schwickerath[31]:

> Melanomas of the choroid, on account of their dark colors, can be destroyed by light coagulation even if they are bigger than a disc diameter but not too elevated. In cases of the larger melanomas the combination of Weve's diathermy treatment with light coagulation can be applied. . . .
>
> We start with a coagulation around the tumor to prevent detachment. Afterwards the tumor itself can be coagulated. Experience has taught us not to try to destroy a big tumor in one session. We therefore perform the coagulation gradually up to 50 times to prevent vascular disturbance. Even tumors lying beside the disc or macula can be destroyed by our method.

In a recent communication Dr. Meyer-Schwickerath reports on ten patients with small tumors successfully treated. The longest period of follow-up is five years.

As in all of these problems, validity of the results in treating malignant melanoma depends entirely upon the accuracy of clinical diagnosis, since in no instance was the diagnosis of malignant melanoma confirmed histologically. Certainly final evaluation of the heliocautery in the treatment of uveal tumors must await more extensive application, and one should be skeptical about its value in larger tumors.

28. *Dr. Leopold, after enucleation has been done, do you think the presence of extrascleral extension necessarily gives a poor prognosis?*

DR. LEOPOLD: Until very recently it was felt that if the extrascleral extension is well encapsulated and has not been broken into by dissection at the time of enucleation, the prognosis may not be any worse than enucleation in which the tumor is well contained within the globe.

However, Dr. Zimmerman and his coworkers have analyzed the data in the Armed Forces Institute of Pathology and have discovered 95 cases in which there was extraocular extension large enough to be seen by the naked eye or which, even though microscopic in size, had obviously been cut through by the surgeon during the enucleation. In only 22 cases did orbital recurrence ensue (approximately 23 per cent). On the the other hand, 75 persons (79 per cent) died of metastatic disease and five others died of unknown cause. Of the 75 tumor deaths, 63 occurred within five years of enucleation.

Thus, it is the opinion of the members of this symposium that the presence of extraocular extension of the type described above is an indication of poor prognosis, as one out of every four patients will have orbital recurrence and four out of five will die from metastatic disease.

29. *If extrascleral extension is found, would you do an exenteration?*

DR. LEOPOLD: Most ophthalmologists have been guided by whether or not the extrascleral extension was encapsulated. If it is encapsulated and not broken into by the operative procedure, enucleation with implant may prove adequate.

Orbital recurrence after enucleation of an eye containing intraocular melanoma is usually the consequence of extraocular extension of the tumor and incomplete surgical extirpation of the malignant tissue. With this in mind, it would appear that immediate exenteration should be considered whenever extraocular extension is nonencapsulated, is extensive, necrotic or obviously disturbed by the enucleation procedure. It should also be performed when microscopic study has shown that the extraocular extension has been cut through by the surgeon during the enucleation.

Results with exenteration do not prove unequivocally that the patients have benefited from such procedure. It may be that the life of the patient is prolonged somewhat, but this is difficult to establish. Nevertheless, on the basis of our present evidence, which admittedly is not clear cut, the exenteration is recommended.

30. *How would you manage a case with recurrence in the orbit several months after enucleation?*

DR. LEOPOLD: Patients vary in their ability to tolerate orbital recurrences. The recurrence may not appear for many years after the initial enucleation, suggesting that the tumor cells have been there but for some reason have been dormant and/or grew extremely slowly. The patient may have some delicately balanced resistance to the growth of this orbital recurrence, as well as of similar tissue elsewhere in the body; this might be disturbed by removal of the orbital contents. This is pure conjecture. Therefore, it would seem advisable that an exenteration be performed when the recurrence is discovered, provided that there is no evidence of metastatic lesions elsewhere in the body. The results after such exenteration are not impressive. Reese reported 100 per cent mortality in eight cases that he had seen. Naquin,[33] in 1954, reported only one individual out of nine who survived over five years after exenteration for orbital extensions of choroidal melanomas.

McRae (1953) has described one patient who survived thirteen years after exenteration for orbital recurrence of a malignant melanoma, but of the last three patients with orbital recurrence seen at Wills Hospital, two were dead within six months after exenteration.

Dr. Zimmerman at the Armed Forces Institute of Pathology has found that of eight patients with orbital recurrence

who received no therapy and could be followed, six were dead in less than one year. The average duration was fifteen months.

31. *Dr. Zimmerman, what morphologic factors are related to prognosis?*

DR. ZIMMERMAN: Dr. Leopold has already discussed the significance of intraocular extension. Evidence that an orbital extension has been incompletely removed by the surgeon may not be obtained except by microscopic examination. Sometimes massive invasion of vortex veins is noted by the surgeon at the operating table, but more often this, too, is detected only after histologic study. I believe that such vascular invasion is of grave prognostic significance.

TABLE II*

PROGNOSIS FOR 1,624 PATIENTS WITH MALIGNANT MELANOMA OF THE CHOROID AND CILIARY BODY

CELL TYPE	5 YR. MORTALITY PERCENTAGE OF TUMOR DEATHS
Spindle A	5
Spindle B	13
Fascicular	20
Necrotic	45
Mixed	52
Epithelioid	69

*Zimmerman, Paul, and Parnell.[57]

TABLE III*

PROGNOSIS FOR 1,624 PATIENTS WITH MALIGNANT MELANOMA OF THE CHOROID AND CILIARY BODY

CELL TYPE	5 YR. MORTALITY PERCENTAGE OF TUMOR DEATHS
Spindle A	5
Spindle B and fascicular	14
Mixed and necrotic	51
Epithelioid	69

*Zimmerman, Paul, and Parnell.[57]

TABLE IV*

PROGNOSIS FOR 1,624 PATIENTS WITH MALIGNANT MELANOMA OF THE CHOROID AND CILIARY BODY

FIBER CONTENT	5 YR. MORTALITY PERCENTAGE OF TUMOR DEATHS
Heavy	6
Marked	19
Medium	31
Light	41
Absent	51

*Zimmerman, Paul, and Parnell.[57]

TABLE V*

PROGNOSIS FOR 1,624 PATIENTS WITH MALIGNANT MELANOMA OF THE CHOROID AND CILIARY BODY

PIGMENT	5 YR. MORTALITY PERCENTAGE OF TUMOR DEATHS
Light	21
Medium	35
Marked	43
Heavy	45

*Zimmerman, Paul, and Parnell.[57]

Callender's histologic classification of intraocular malignant melanomas is still in active use at the Armed Forces Institute of Pathology, for it has given reliable information concerning prognosis. This classification is based on three cell types: "spindle A," "spindle B," and "epithelioid." In addition, there is a variety of spindle cell melanoma characterized by palisading of the nuclei of tumor cells. This is called the "fascicular" type. Those tumors containing mixtures of spindle and epithelioid cells are classified as "mixed." Some tumors are too necrotic to be classified otherwise. These melanoma types are listed in order of increasing malignancy in table II.

I believe this classification can be simplified without seriously affecting its usefulness. Pure spindle A melanomas are uncommon, and they almost always behave as benign lesions. Fascicular tumors are composed of spindle B cells, and their prognosis does not differ significantly from that of other spindle B tumors, so these two types may be lumped together. For the same reason, we may place mixed and necrotic tumors in the same category. Thus we come up with the simplified classification presented in table III.

Other morphologic factors of established prognostic significance include the degree of reticulum fiber deposition (table IV) and the amount of pigment production (table V).[55,57]

32. *Dr. Reese, has tissue culture yielded any important information about the behavior of melanomas?*

DR. REESE: Cultures of uveal melanomas show a growth ranging from a branching polygonal-shaped pigment-bearing cell to a long, bipolar spindle-shaped cell with or without pigment. These latter cells have the characteristics of Schwann cells. All gradations between these two extremes can be found. Regardless of the cell type of the tumor cultured, there is transition from one type of cell to the other. Therefore, just as there are two principal cell types seen in conventional histologic studies, so there are the same two types observed in cultures. The presence of more than one cell type in the same tumor is, of course, consistent with findings in other tumors, especially those from the neuroectoderm. As uveal melanomas seem to stem from the neural crest, it is not surprising that their cells are pluripotential.

SUMMARY AND CONCLUSIONS

DR. DUNPHY: It now becomes my province to summarize what the various members of the panel have said and to emphasize certain points. From what has been told us this morning, it is apparent that the diagnosis and management of intraocular melanomas may pose many problems.

Dr. Reese and Dr. Zimmerman have mentioned a number of lesions that have been mistaken for malignant melanomas of the uveal tract. From a clinical point of view, Dr. Reese has listed inflammatory lesions, macular degenerations, benign melanomas, metastatic carcinomas and serous detachments of the retina as those most frequently confused with malignant disease of the choroid. From his pathologic experience, Dr. Zimmerman states that disciform degenerations of the macula, hemorrhagic detachments of the retina and separation of the choroid are often found in eyes enucleated for malignant melanoma of the choroid.

In diagnosing tumors of the iris and ciliary body, Dr. Reese has cited a number of other lesions which must be considered in the differential diagnosis, such as intraepithelial cysts, granulomas, and leiomyomas. Dr. Zimmerman has reported on a wide variety of lesions found in eyes which were enucleated because of a mistaken diagnosis of malignant melanoma of the iris. Among these are discrete lesions, such as staphylomas, anterior and posterior chamber cysts, granulomas, nevi, hemangiomas and leiomyomas; also, diffuse processes leading to an alteration in the pigmentary characteristics of the iris.

Among the various diagnostic signs, Dr. Forrest has mentioned the presence of certain external abnormalities, such as melanosis oculi, dilated episcleral vessels and increased resistance of the sclera over the lesion to palpation with a blunt instrument. These signs are not frequent but, when present, are corroborative in some doubtful cases.

Slit lamp examination and gonioscopy are, of course, indispensable for the study of suspected lesions of the iris.

The ophthalmoscope can be used to great effect in the diagnosis of malignant melanomas of the choroid. Dr. Reese has emphasized its important function in retroillumination of a suspicious lesion. He also mentioned the abnormal vascular patterns which may be found over the surface of the lesion and the occurrence of light brown pigmentation in tumors, in contrast to the jet black pigment seen in inflammatory lesions.

Ordinary transillumination, as Dr. Leopold has told us, is not always reliable and, unless the transilluminating source is directly behind the lesion, it is useless.

The uptake of radioactive phosphorus by malignant melanomas is an established fact. However, as Dr. Leopold has pointed out, there are important limitations to the clinical effectiveness of this test. It must be properly performed and finds its greatest usefulness in lesions accessible to the small Geiger counter. The thing to remember is that a negative test means nothing, whereas a positive test is of real significance in doubtful cases. Dr. Leopold has also warned us about some of the hazards in using radioactive phosphorus.

Apparently the use of ultrasonic waves to differentiate malignant melanomas from serous detachments of the retina has not yet reached a stage of development to be practical.

Except for iris lesions, biopsy is generally considered a hazardous procedure; Dr. Forrest has given us the reasons why. Aspiration and study of the subretinal fluid in detachments associated with suspicious lesions is, by contrast, relatively innocuous. However, in the small lesions, which usually constitute the diagnostic problems, cells are not likely to be found in the subretinal fluid, since in these cases the tumor remains within the lamina vitrea. In any case, the differentiation of tumor cells from pigmented histiocytes would be uncertain. Therefore, it is concluded that the test is probably not reliable enough to justify its use routinely.

Dr. Zimmerman, in reviewing his own experience and that of others, tells us that about 10 per cent of intraocular malignant melanomas are not suspected clinically before enucleation. He points out the possibility that the tumor itself, because of necrosis, may give rise to the complications which lead to opacification of the media. This brings up the question whether all blind eyes should be removed on the chance that they might harbor malignant melanomas. Dr. Leopold has expressed the opinion of the panel that enucleation should be performed only in those blind eyes in which the cause of the blindness cannot be established, in which the fundus cannot be examined, or in which there is some other reason for enucleation besides the remote possibility of a tumor being present.

Assuming now that a diagnosis of intraocular malignant melanoma has been made by the various tests at our disposal, what shall be our course of action? Immediate enucleation or watchful waiting? Dr. Zimmerman has warned against considering all clinically diagnosed uveal melanomas as surgical emergencies, particularly small choroidal tumors and the great majority of all iris tumors. Although the cell type of the tumor definitely influences the prognosis, this information is not available to the surgeon beforehand. However, the size of the tumor, as seen clinically, is important; the best prognosis being in the small, slow-growing lesions, which are usually of the spindle-cell type and rarely metastasize. Therefore, it is felt

that small questionable fundus lesions should be followed with serial photography and visual field studies until there is absolutely no doubt of their progression. In considering iris tumors, it is comforting to learn that in Dr. Zimmerman's series of proved iris melanomas, metastasis was extremely rare.

When enucleation is performed, the presence of visible extrascleral metastatic lesions always presents a problem. Dr. Leopold has expressed the feeling of this group that if the extension is well encapsulated and not broken into by the surgical procedure, simple enucleation with implant is justified. If, however, it is not encapsulated, it is better to do an exenteration forthwith, although it is difficult to say whether the patient will be benefited, since the results of exenteration are not too good. If recurrence takes place in the orbit several months after enucleation, the prognosis is definitely bad. Occasionally one hears of a cure by further surgical intervention and even irradiation, but most of these patients die as the result of metastasis within five years.

We have considered the other methods of attacking intraocular malignant melanomas besides enucleation. Coagulation of the tumor with diathermy has recently received some attention, particularly in one-eyed individuals for whom enucleation is almost unthinkable. It is the consensus of the panel that this is probably not a worth-while procedure and may be fraught with considerable danger if perforating diathermy is used, because of dissemination of tumor cells into the orbit. In case of large tumors this perforating diathermy would probably be necessary in order to reach the lesion. Dr. Reese rightly points out that with this method of treatment one cannot be sure what he is treating; hence, reported successes are open to serious doubt. In case of smaller tumors it would be justifiable to wait, since the chances of metastasis would not be great anyway.

In regard to irradiation, we feel that this is not to be considered as a method of treatment of malignant melanomas. Dr. Forrest has emphasized that this type of tumor is not radiosensitive. Reported successes in the literature are infrequent, and in none of them has there been histologic confirmation of the diagnosis.

Dr. Forrest has also explored the field of chemotherapy. Although the nitrogen mustards and their analogues are useful in certain tumors, such as retinoblastoma, they apparently do not effect any permanent cure of melanomas.

We feel that the claims of Meyer-Schwickerath that the use of heliocautery can destroy malignant melanomas of the uveal tract must await further confirmation. The idea is most interesting but, here again, the reported cures are without histologic corroboration.

Finally, we feel that there remains a lot to be learned about the behavior of malignant melanomas of the uveal tract. This is often unpredictable in contrast to that of malignant melanomas of the skin, where a rapid downhill course is usual. We all know that some patients who refuse enucleation go on for years in perfect health with no sign of metastasis, while in others metastasis occurs in spite of early enucleation. Is it possible that early metastasis actually occurs in all cases but that in certain ones the tumor cells are restrained from growing in their new environment by some factor as yet unknown? In other words, do we actually save life by enucleation? May it not already be too late? No one knows for sure; until we have more knowledge on the subject, we must assume that we do and act accordingly.

At this point I would like to make a plea that every ophthalmologist make a special point to follow up his cases in which enucleation is not done and see

what happens to them. Perhaps the American Academy of Ophthalmology and Otolaryngology might establish a registry to which such cases could be referred for periodic evaluation and follow-up. It is our hope that this will be done, for only by such a study can we get an answer to this perplexing question of whether or not enucleation really prolongs life expectancy.

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THE PATHOLOGY OF THE TRABECULAR MESHWORK IN PRIMARY OPEN ANGLE GLAUCOMA

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PRIMARY open angle glaucoma is associated with an increased resistance to aqueous outflow. This increase in resistance is organic in nature, for it has been shown by perfusion studies[4,14,17] that it is essentially the same after the eye is enucleated as before. There is no doubt that this higher resistance to aqueous outflow lies in the area of the filtration angle, but its exact site is still disputed.

Only a small number of eyes with early open angle glaucoma have been described histologically because of the difficulty of obtaining specimens before synechias and other late changes have occurred. However, a number of workers have described pathologic changes in the trabecular meshwork of such eyes.

In pathologic studies of four enucleated glaucomatous eyes, Teng, Paton and Katzin[28] found degenerative changes in the entire area of the drainage angle, but they were most marked in the external part of the trabecular meshwork. These changes consisted of degeneration of collagen tissue and proliferation of the endothelium of Schlemm's canal. In their studies, they found similar degenerative changes in 10 per cent of eyes from the eye bank of people over 50 years of age; but these pathologic alterations were never seen in eyes of people under the age of 20. They suggested that open angle glaucoma is due to a primary degeneration in the connective tissue of the filtration angle.

In another study of eyes with primary open angle glaucoma, Theobald and Kirk[29] found thickening and sclerosis of the trabeculae and collector channels in the sclera. In some of their illustrations they noted narrowing of Schlemm's canal and a spongy edema of the external trabeculae, with deposition of pigment in this area. Their studies led them to believe that hypertrophy and sclerosis of collagenous fibers of the sclera associated with aging compresses and narrows intrascleral veins, producing glaucoma.

Maggiore,[24] Francois and his colleagues,[13] and others have found thickening and sclerosis of the trabecular fibers in cases of primary glaucoma.

Ashton, Brini and Smith[1] found artefactual adhesions in Schlemm's canal in 25 per cent of normal eyes. They warn against misinterpreting as pathologic, variations in the microscopic anatomy of the meshwork which may be found in normal eyes.

That the trabecular meshwork is the area producing the obstruction has been advocated by some writers.[16,23] It has recently been pointed out that the anatomy of the meshwork is incompletely visualized in ordinary pathologic studies because the meridional sections which are made allow only an unsatisfactory view of the meshwork.[19] Transverse and, particularly, tangential sections of

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the meshwork are necessary to appreciate its anatomy fully.

Believing that such sections are equally essential for a study of the pathologic changes in the trabeculae, a study has been made in transverse, meridional and serial tangential sections of eyes with primary open angle glaucoma, and of eyes with a variety of types of secondary glaucoma with open filtration angles.

Striking degenerative changes were found in the trabecular meshwork of eyes with primary and with secondary glaucoma. The character of these changes suggests a new conception of the pathogenesis of primary open angle glaucoma.

MATERIALS AND METHODS

The trabecular meshwork of 16 eyes with primary open angle glaucoma and of 11 eyes with a variety of types of secondary glaucoma with open filtration angles were studied.

Most of the material was obtained from the Armed Forces Institute of Pathology, which cooperated in the study by forwarding to me unused wet tissue from eyes with open angle glaucoma.

The material forwarded by the Armed Forces Institute of Pathology consisted of one or more meridional sections of each eye and the remaining wet tissue of the eye not used in making the original block. From this tissue enough material was usually obtained to prepare tangential sections of the meshwork *in situ* from three different quadrants of the eye.

In every case meridional sections as well as tangential sections were studied.

The tangential sections were prepared in a manner described in a previous paper.[9] Because of the unavoidable slight curvature occurring in the tissue studied, oblique and transverse sections of the trabecular meshwork could be seen in some of the sections in almost every eye. Alternating slides of the flat sections were stained with Masson's trichrome stain. The remainder of the slides were stained with a variety of stains, including Verhoeff's elastic tissue stain, hematoxylin and eosin, Mollier's elastic, and periodic acid-Schiff. The Masson stain was found to be particularly helpful. Sclerosis and an increase in collagen are strikingly demonstrated with this stain in a deep blue color. The nuclei and other components of the meshwork also show up well with the Masson's stain. From 10 to 150 sections of the trabecular meshwork were examined in each glaucomatous eye, a total of over 2,000 sections of the trabecular meshwork of glaucomatous eyes. These sections were compared with similar sections made from normal eyes reported previously.[9]

All of the cases of both primary and secondary glaucoma were characterized by open angles. The histories, as given, were carefully studied (table I). In twelve of the 16 eyes with primary open angle glaucoma, the opposite eye was known to be involved by the same disease. In the other four cases, either the condition of the other eye was unknown or it was said to be uninvolved. In these cases, however, when the rest of the history of the disease was considered along with the pathologic sections showing the open angles, deep chamber and cupped discs, there was little room for doubt of the diagnosis of primary open angle glaucoma.

Some of the eyes were completely blind before they were removed; others were removed much earlier in the course of the disease. In one case, a 69-year-old woman with early primary open angle glaucoma and small Bjerrum scotomas, the eyes were removed for study immediately after her death from multiple myeloma.

The cases of secondary glaucoma all evidenced open filtration angles with no anterior or posterior synechias. The di-

TABLE I

LIST OF CASES: PRIMARY OPEN ANGLE GLAUCOMA

NO.	SOURCE	PERTINENT HISTORY	AGE	TENSION	OTHER EYE
1 & 2	Stanford Eye Bank	Gradual blurring of vision. Occasional morning headaches. Tension frequently in mid-thirties, but usually well controlled with pilocarpine. Bilateral small Bjerrum scotomas. Vision 20/30 both eyes. Incipient cataracts. Died of multiple myeloma.	69	35	Primary open angle glaucoma, both eyes
3	AFIP 499450 Right eye	Right eye began to lose vision 4 years prior to enucleation. No light perception.	69	40+	Chronic simple glaucoma
4	AFIP 499450 Left eye	Left eye began to fail 5 years prior to enucleation. Blind.	69		Chronic simple glaucoma
5	Stanford Eye Path. 56-10	Poor vision 1 year. Painful 1 week. Recent hyphemia. No light perception.		114	Glaucoma simplex; tension, 43
6	AFIP 686098	Blind painful left eye for 8 months.	55	Stony hard	Chronic simple glaucoma controlled with miotics
7	AFIP 720864	Primary open angle glaucoma, 23 years duration. Pilocarpine 20 years. Visual field constricted 18 years. Tension elevated 5 years. No vision.	76	100 (McLean)	Chronic simple glaucoma
8	AFIP 577292	Loss of vision 6 years. Pain 4 weeks.	?	60+	Primary open angle glaucoma
9	AFIP 638508	Gradual loss of vision 13 years. Optic nerve atrophic.	72	100	Blind of chronic glaucoma
10	AFIP 732016	Eye had been blind for 20 years.	69	53	Chronic open angle glaucoma; tension, 32
11	Stanford Eye Path.	Loss of vision 1 year. No light perception.	84	70	Primary open angle glaucoma; iridencleisis
12	AFIP 260484	Tension elevated in both eyes. Field constricted to 10°, pain 3 weeks. Light perception.	56	60	Chronic glaucoma; enlargement of blind spot; tension, 40
13	AFIP 684933	One sister has glaucoma. Glaucoma; deep chamber; complete glaucomatous cupping. Branch occlusion of central retinal vein	71	60	Small central cupping; tension, 22

TABLE I (continued)

NO.	SOURCE	PERTINENT HISTORY	AGE	TENSION	OTHER EYE
14	AFIP 699279	No history of injury. Recent severe pain. Duration of blindness unknown. Deep cupping of disc. Open angles.	73	100	Vision, finger counting. Macular scar? Tension, 23
15	AFIP 586616	No history of injury. Glaucoma many years. Corneal ulcer and pain for 3 weeks. Deep cupping of disc. Open angles		65	Normal?
16	AFIP 270786	Pain for 1 week. Absolute glaucoma. Complete cupping of disc. Open filtration angles.	78	90	Apparently normal at time affected eye was enucleated

agnostic data regarding the patients with secondary glaucoma are listed in table II. The average age of these patients was much lower than the average age of the patients with primary glaucoma. In many of the cases the original inciting cause (blunt injury, chorioretinitis, hemorrhage, etc.) had subsided years before; but the glaucoma continued and progressed until enucleation was necessary.

Three patients with glaucoma of doubtful origin with Descemet's membrane extending over the filtration angle were examined but were not included in this study because it was felt that the profound degeneration found in the trabecular meshwork in these cases was not the subject of the present investigation.

Two simple experiments, which will be described later, were carried out on eyes from the Stanford Eye Bank to help elucidate the meaning of some of the histologic variations noted.

RESULTS

The study of sections of the trabecular meshwork in every plane reveals the nature and extent of the degenerative lesions much more clearly than the study of sections in the meridional plane alone. In most instances the tangential and transverse sections are particularly revealing; but in others, the meridional sections furnish information which cannot be obtained in any other way.

Striking pathologic changes are noted in the trabecular meshwork of eyes with primary open angle glaucoma (figs. 1-9). These changes are degenerative in nature. They begin and are most marked on the external part of the trabecular meshwork or the inner wall of Schlemm's canal. The extent of the degeneration varies markedly in different areas of the limbus. In a given eye the meshwork may appear almost normal at 2 o'clock and show severe degeneration at 4 o'clock. In these respects, (1) the presence of degenerative changes in the trabecular meshwork and (2) the site of these changes in the meshwork, the work of Teng, Paton and Katzin[28] is confirmed. Degenerative changes in the collector channels were not as marked or as consistent as the trabecular changes.

Thickening of the trabecular beams and sclerosis of the trabeculae, as noted by Theobald and Kirk,[29] is seen in most of these eyes but is not invariably present. These changes are not marked in the least advanced cases.

The interlamellar stenosis reported by Francois and his colleagues[14] is fre-

TABLE II

SECONDARY GLAUCOMA WITH OPEN FILTRATION ANGLES

NO.	CASES	DIAGNOSIS	HISTORY	AGE	RACE	OTHER EYE	TENSION
1	AFIP 724563	Glaucoma secondary to contusion.	Blunt injury 7 years prior to enucleation. Eye blind for 4 years; hard and painful for 2 weeks.	37	N	Normal	Hard
2	AFIP 680536	Glaucoma secondary to trauma	Injury while hammering. Cataract and secondary glaucoma.	36	W	Normal	65
3	AFIP 636508	Glaucoma secondary, cause and type undetermined	Corneal leukoma; keratitis; progressive loss of vision; light perception only	35	N	Normal	Hard
4	AFIP 654466	Glaucoma secondary to old chorio-retinitis; possible toxoplasmosis	Severe old chorioretinitis scarring; pain for 3 weeks.	63	W	Normal	40
5	AFIP 543663	Glaucoma secondary to old endophthal-mitis	Recurrent attacks of pain for 15 years; secondary cataract.	?	W	Normal	
6	AFIP 716059	Glaucoma secondary to injury and dislocated lens	Blind since injury. Enucleation because of endophthalmitis and sclerosing keratitis.	29	W		
7	AFIP 759801	Glaucoma secondary to hemangioma of the choroid (Sturge-Weber syndrome)	Blind painful left eye, glaucomatous since early childhood.	42	W	Normal	
8	AFIP 672943	Secondary glaucoma cataract	Senile cataract in other eye. Affected eye blind.	77	W	Normal tension	Hard

TABLE II (continued)

NO.	CASES	HISTORY	DIAGNOSIS	AGE	RACE	OTHER EYE	TENSION
9	AFIP 697467	Aphakia, glaucoma	Onset of glaucoma after cataract extraction. Iridectomy 3 years later failed to control glaucoma. Eye blind.	89	W		Hard
10	666 Episcopal EENT Hosp. Wash., D. C. Contribution of Dr. Benjamin Reeves	Glaucoma secondary to central vein thrombosis	A fibrovascular membrane is not present on the iris. No synechias. Marked pigmentation of the iris due to hemorrhage and which stains for iron.	55	N	Normal	56
11	AFIP 502446	Glaucoma secondary to intraocular foreign body and siderosis bulbi.	Dislocated cataractous lens. Keratitis.	41	W	Normal	Stony Hard

quently seen but is believed to be merely a change due to the immediate effect of increased intraocular pressure. The newly formed blood vessels in the region of Schlemm's canal which they report in one case of secondary glaucoma and call "rubeosis" of the trabeculae is found in this study in cases of primary and secondary glaucoma.

The pathologic changes seen in the trabecular meshwork and Schlemm's canal in these eyes with primary open angle glaucoma are listed below. All of these changes were not noted in every eye but some were seen in each eye.

1. Narrowing and partial obliteration of Schlemm's canal.
2. A thickening and foamy appearance of the tissue just beneath Schlemm's canal (the external part of the trabeculae, the "pore area").
3. Frequently an increase in the number of cells on the inner wall. Most of the new cells are probably fibroblasts. Occasionally a few inflammatory cells are seen. In many instances with marked fibrosis, there is a decrease in the number of nuclei in the trabeculae. The cells seen in the meshwork are frequently less plump and less uniform in appearance.
4. Narrowing of the interlamellar spaces, as seen in routine section with the nuclei assuming a flattened appearance.
5. Fibrosis and adhesions in Schlemm's canal.

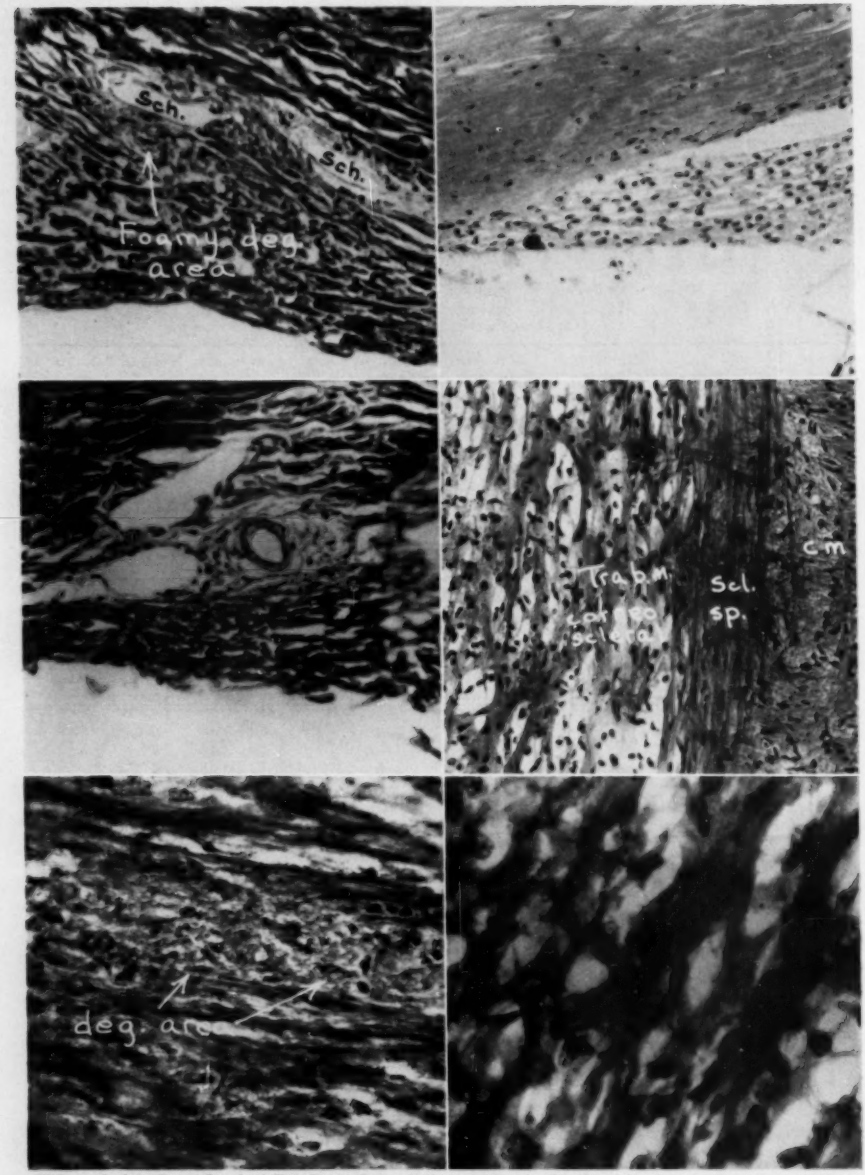

FIG. 1 (Left)—From Stanford Eye Bank. Eyes of 69-year-old white female with primary open angle glaucoma, both eyes. Tension 25 to 35 mm. Hg in both eyes with pilocarpine; small Bjerrum scotoma in each eye. Vision 20/30 both eyes; incipient cataracts. Patient died of multiple myeloma. (Top) Meridional section, with Masson's stain. Foamy degenerated appearance of outer part of meshwork. (Center) Meridional section, with Masson's stain. A blood vessel is present in the foamy area near Schlemm's canal. (Bottom) Tangential section; corneoscleral meshwork; Verhoeff's stain. A degenerated area is present.

FIG. 2 (Right)—Eyes from Stanford Eye Bank. Normal meshwork for purposes of comparison. (Top) Meridional. (Center and Bottom) Corneoscleral tangential sections. Note the plump, uniform quality of the trabecular nuclei.

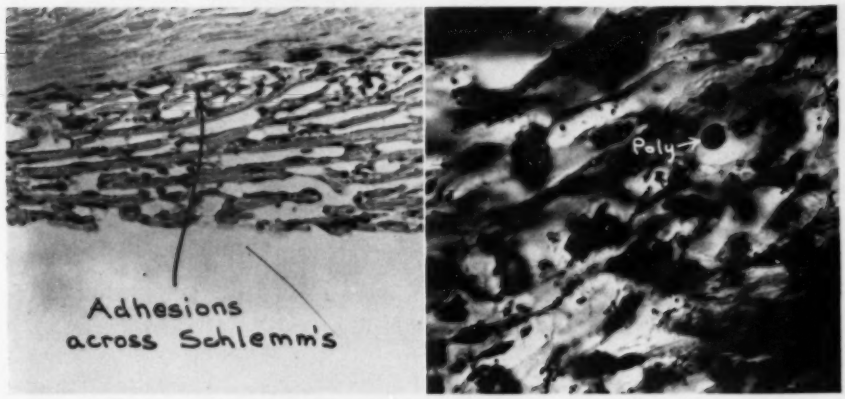

FIG. 3—AFIP 577292. Primary open angle glaucoma in both eyes. Loss of vision for six years. Tension, 60 mm. Hg. (Left) Meridional section. Schlemm's canal is virtually obliterated by adhesions across it. (Right) Tangential section near inner wall. Foamy granular appearance; incraese in pigment; occasional inflammatory cell.

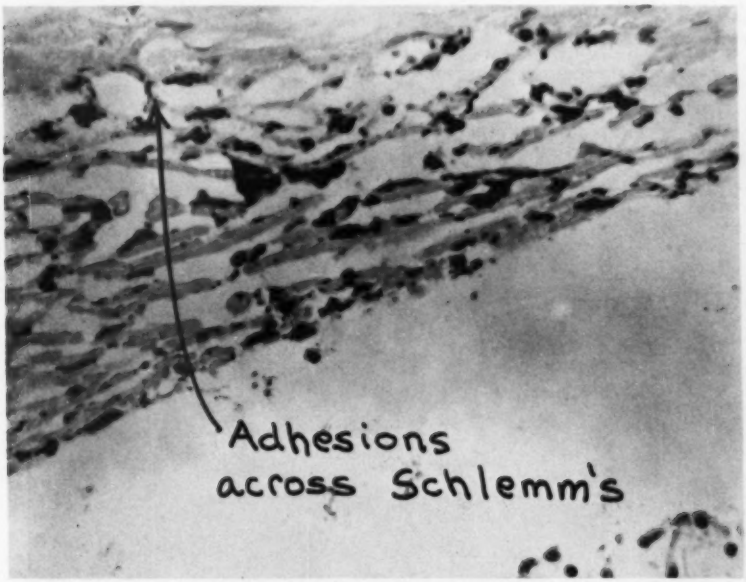

FIG. 4—AFIP 720864. Eye of a patient 76 years of age. Primary open angle glaucoma in both eyes of twenty-three years' duration; pilocarpine therapy for twenty years. Meridional section. Schlemm's canal is virtually obliterated by adhesions across it.

FIG. 5—AFIP 686098. Age of patient, 55 years. Primary open angle glaucoma in both eyes. Tension stony hard. Oblique, almost transverse, section. Neovascularization of inner wall of Schlemm's canal.

FIG. 7—AFIP 586616. Glaucoma of many years duration. Open filtration angles. Cupped disc. Condition of other eye unknown. Dense scarring of trabecular meshwork.

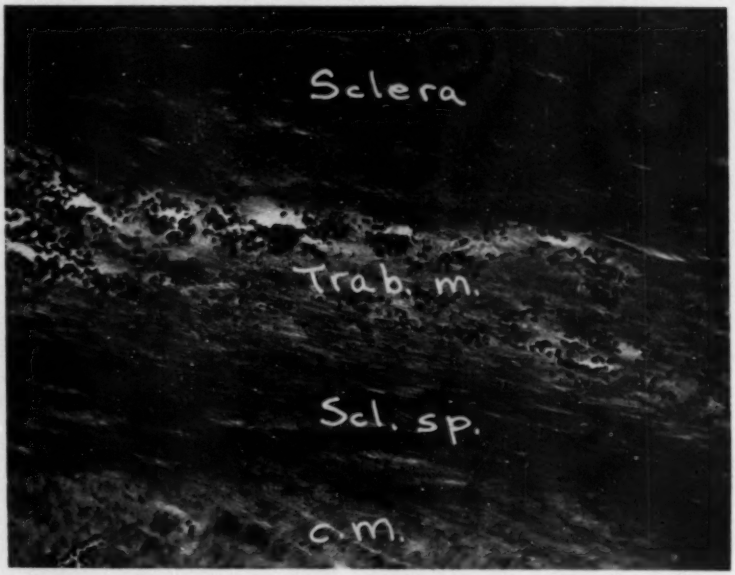

FIG. 6—AFIP 638508. Age of patient, 72 years. Primary open angle glaucoma in both eyes. Gradual loss of vision for thirteen years. Marked degeneration and scarring of trabecular meshwork.

FIG. 8—AFIP 699279. Open angle glaucoma. Tangential section. Large melanophores are seen in the uveal meshwork.

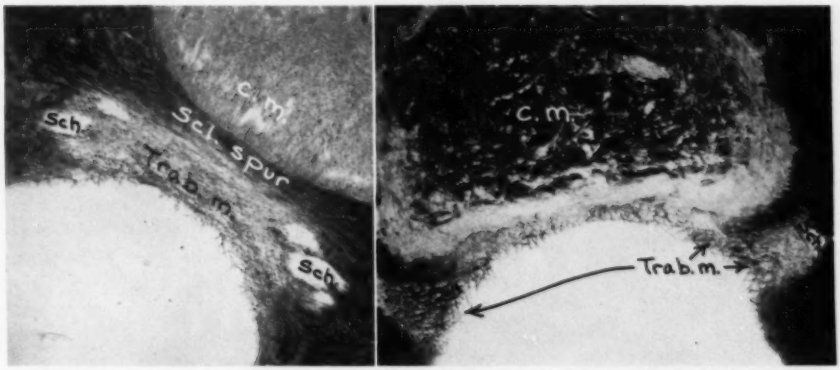

FIG. 9—(Left) AFIP 67294. Eye enucleated because of melanoma of choroid. Normal meshwork. Masson's stain. Curved tangential section for comparison with similar section (Right) AFIP 499450. Age of patient, 69 years. Primary open angle glaucoma. Marked sclerosis of trabecular meshwork and ciliary body.

6. Recanalization of Schlemm's canal by proliferation of endothelium along adhesive bands.
7. Vascularization of this area. Schlemm's canal is sometimes divided into small compartments which are indistinguishable from blood vessels and frequently contain blood.
8. Thickening of uveal and corneoscleral beams with an increase in collagen content in these beams.

FIG. 10—AFIP 636508. 35-year-old Negro man with unilateral secondary glaucoma with open filtration angles. Corneal leukoma. Progressive loss of vision. No synechias. Optic disc cupped. Other eye normal.

9. Hyalinization of the meshwork, particularly the external part.

10. An increase in pigment having a characteristic distribution is usually seen. Pigment having the same distribution is seen in normal eyes of older individuals but is usually more marked in the glaucomatous eyes. The largest part of the pigment is contained in large melanophores or macrophages containing the granules, but some of it is located in the trabecular cells and some of it lies free. Most of it was located either in the uveal meshwork in large melanophores, or in the inner wall of Schlemm's canal, the bulk of the corneoscleral meshwork being comparatively free of pigment.

Some of the less marked changes are sometimes found in normal eyes but the more severe changes such as vascularization, marked fibrosis and hyalinization with complete loss of normal architecture (in tangential section) that are frequently seen in the eyes with primary open angle glaucoma are never seen in normal eyes.

Secondary Glaucoma With Open Filtration Angles

The pathologic findings in the trabecular meshwork of the eyes with secondary glaucoma were quite surprising. Precisely the same types of degeneration that were found in primary open angle glaucoma were seen in the filtration angles of these eyes (figs. 10-12).

(Top) Meridional section. The meshwork is compressed and appears to be pushed anteriorly, obliterating Schlemm's canal. The pigment is heaviest near the inner wall of Schlemm's canal. Compare with figure 13 and figure 14.

(Center) Meridional section opposite angle. New adhesions are forming across Schlemm's canal. An increased number of cells is present in and near Schlemm's canal.

(Bottom) Tangential section. Degenerated area of corneoscleral meshwork containing acute and chronic inflammatory cells.

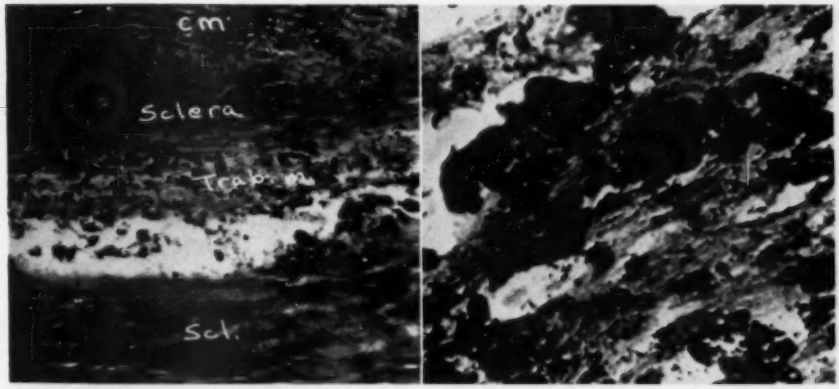

FIG. 11—AFIP 742563. 37-year-old Negro man. Glaucoma secondary to contusion. Blunt injury seven years prior to enucleation. Blind for four years. Painful eye for two weeks. Other eye normal. (Left) Tangential section. There is marked scarring and loss of normal architecture of the corneoscleral meshwork. Pigment deposits are increased. (Right) Tangential section; high power. The number of trabecular cells is decreased.

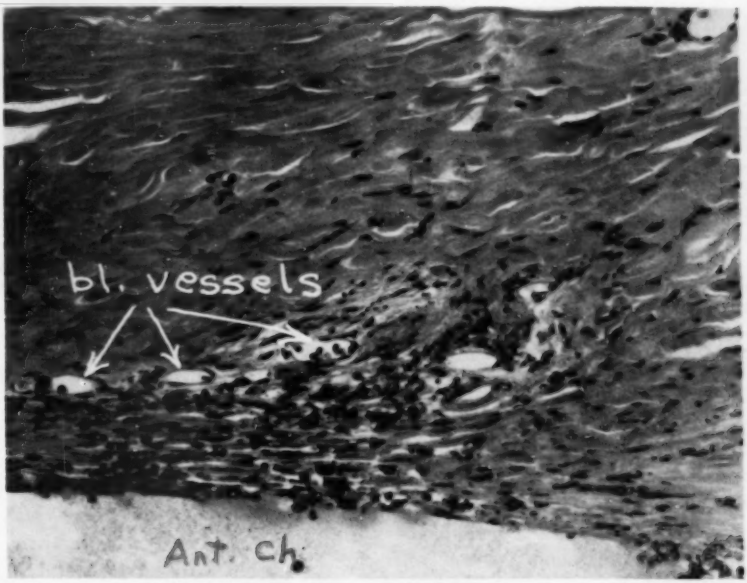

FIG. 12—AFIP 697467. Secondary glaucoma. Onset of unilateral glaucoma three months after cataract extraction. Iridectomy three years later failed to control glaucoma. Meridional section. Canal obliterated by scar tissue containing an increased number of nuclei. Neovascularization of Schlemm's canal.

Although some of these individuals were quite young, sclerosis, degeneration and increase in collagen were marked in almost every case. The meshworks were quite unlike those of normal eyes and every one of the pathologic changes that could be found in primary open angle glaucoma was found in these eyes. The degeneration was, in general, just as extensive in these eyes as those with primary open angle glaucoma. As occurred in the cases with primary open angle glaucoma, some eyes had less extensive changes than others.

What is the meaning of these histologic alterations, some of which are found to be a small degree in eyes of normal older individuals and to a much greater degree in eyes of patients with primary or secondary open angle glaucoma?

The histologic changes may be divided into four groups:

1. Changes due to age.
2. The increase in pigment and its distribution.
3. Changes due to the immediate effects of increased intraocular pressure.
4. Changes due to the effects of prolonged increased intraocular pressure.

1. *Changes Due to Age*

In normal eyes of individuals of advanced age the trabecular beams become thicker and contain more collagen. This is in agreement with findings of Theobald,[29] Verhoeff,[30] Rones[25] and others. Degenerative changes in the external trabeculae, thickening and a foamy appearance of the area are sometimes present. The extensive changes such as vascularization, recanalization or obliteration of Schlemm's canal, marked fibrosis and hyalinization are not found normally.

2. *The Increase in Pigment and Its Distribution*

The distribution of pigment in the meshwork is evidence that the smallest openings or chief resistance to the passage of pigment from the anterior chamber to the outside of the eye through the filtration angle is in the region of the inner wall of Schlemm's canal, the "pore area" (fig. 13). The larger cells and clumps of pigment are stopped at the uveal meshwork, but most of the pigment collects near the inner wall of Schlemm's canal, in the "pore area," the great bulk of the corneoscleral meshwork being left relatively free of pigment. It is evident that the pigment has difficulty in getting through this area because of the relatively smaller openings in the meshwork.

FIG. 13—Eye from eye bank. Oblique section. India ink solution made to flow through the eye by injection into the anterior chamber of eye bank eyes tends to collect near the inner wall of Schlemm's canal, indicating that the openings in the meshwork are smallest here. Compare with figure 10 (Top).

By injecting diluted India ink, the same distribution of ink has been produced in our laboratory in eyes from the eye bank, the larger clumps of pigment being caught in the uveal meshwork and the bulk of it being stopped near the inner wall of the canal. Very little pigment or India ink remains in the vessels distal to Schlemm's canal, for it is clear that if the particles are small enough to get through into Schlemm's canal, they have relatively clear sailing from then on. This is not surprising, for we know that the caliber of the vessels between the outer surface of the eyeball and Schlemm's canal is large enough to allow red blood cells to pass into Schlemm's canal, but that the red cells cannot pass through the wall of Schlemm's canal into the anterior chamber.

In addition, the openings of the uveal and corneoscleral meshworks as seen in tangential section are larger than a red blood cell until the region of the inner wall of Schlemm's canal is reached.[9] Here the openings are smaller and hard to see. Huggert[20] and Francois and his coworkers[13] in separate perfusion experiments demonstrated that the size of the smallest openings, the "pore size," is about 2 or 3 microns.

The fact that the amount of pigment increases moderately with age but increased greatly in primary and secondary open angle glaucoma suggests that the daily variations in pressure, and particularly the increase in pressure, dislodges pigment and pigment-bearing cells from the iris, which are then caught and stopped in the trabecular meshwork.

The increase in pigment in the meshwork is certainly not due to aging alone. We have an infant's eye with congenital glaucoma in which tangential sections of the meshwork show a marked increase in the amount of pigment in the trabeculae.

3. *Changes Due to the Immediate Effects of Increased Intraocular Pressure*

When the pressure in the anterior chamber of the eye is increased, the trabecular meshwork is pushed forward (or stretched) compressing the trabecular lamellae and narrowing Schlemm's canal. These findings were present in almost every one of the glaucomatous eyes. This forward displacement (or stretching) and compression of the meshwork is due simply to increased pressure. That was shown in an experiment suggested to me by Dr. Otto Barkan. Desiring to see the histologic appearance of the trabeculae when the anterior chamber is deepened, he suggested that a warm solution of gelatin be injected into the anterior chamber of an eye from the eye bank under a slight pressure. Immediately after the gelatin is injected, it cools and solidifies in the anterior chamber, keeping it deep and under increased pressure. When the eye is fixed and examined histologically the trabecular lamellae are seen to be compressed and pushed forward (or stretched), narrowing Schlemm's canal, just as is found in the majority of glaucomatous eyes (fig. 14).

The forward displacement of the trabeculae with narrowing of Schlemm's canal found in glaucomatous eyes is further evidence that the site of obstruction to aqueous outflow is in the trabeculae near the inner wall of Schlemm's canal. If the chief obstruction to outflow were in the sclera, distal to Schlemm's canal, one would expect Schlemm's canal to be dilated instead of narrowed, but this is never the case. Indeed, the histologic, experimental and mathematical evidence that the chief obstruction to aqueous outflow is on the proximal rather than distal side of Schlemm's canal is now overwhelming,[2,10,16,23] but will not be discussed in detail at this time.

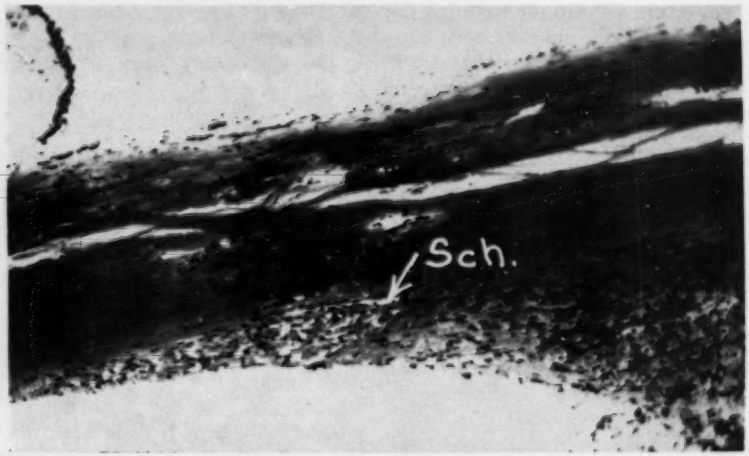

FIG. 14—Eye from eye bank. Pressure in anterior chamber increased by the injection of gelatin. The trabecular meshwork is compressed, narrowing Schlemm's canal.

The nuclei of the trabecular cells appear elongated and compressed in almost all of the glaucomatous eyes but whether this is due to the immediate or to the prolonged effect of increased intraocular pressure is not yet certain.

4. *Changes Due to the Prolonged Effects of Increased Intraocular Pressure*

Let us first consider the cases of secondary glaucoma with open filtration angles. These cases have diverse causative factors. The original increase in intraocular pressure in some cases may have been produced by blockage of the outflow channels by hemorrhage, inflammation, pigment, or intraocular foreign body; or for mechanical reasons such as in intumescent cataract. The tension in many of these cases subsided for a time after the initial bout of glaucoma and then gradually rose to the point where enucleation was done. In all of these cases essentially the same lesions of the trabeculae are seen, but they are in various stages of development in different parts of the meshwork and in different eyes.

First there is an edema or foamy appearance of the inner wall of Schlemm's canal, with an increased number of nuclei in this area. With the canal narrowed and this degenerative process going on in the inner wall, adhesions develop between the inner and outer wall of Schlemm's canal, dividing the canal into compartments with what appears to be new blood vessels or attempts at recanalization of the canal. Later the number of nuclei in the external trabeculae decreases as fibrosis, hyalinization and thickening of the trabecular beams are seen.

The only two things that all of these cases have in common are increased intraocular pressure and degenerative changes in the filtration angle. It is quite clear that these degenerative changes are a result of the prolonged increased intraocular pressure.

In primary open angle glaucoma, precisely the same degenerative changes are seen in the trabecular meshwork; here,

too, these changes must be due to an intraocular pressure high enough to cause trabecular damage.

THE PATHOGENESIS OF PRIMARY OPEN ANGLE GLAUCOMA

The degenerative changes seen in the external trabeculae and Schlemm's canal in cases of primary open angle glaucoma are evidently the cause of the reduced facility of outflow. We have seen that this type of trabecular damage is produced in cases of secondary glaucoma with open angles by prolonged increased intraocular pressure.

In eyes destined to have primary open angle glaucoma, the tension at first is not high. What, then, causes the original trabecular change? Pathologic and clinical evidence combine to suggest the answer to this question.

One of the most characteristic findings of primary open angle glaucoma is a failure of the pressure regulating mechanism. The diurnal variations in intraocular pressure are believed to be due to variations in the production of aqueous. One of the earliest manifestations of primary open angle glaucoma is an increase in the diurnal variations of the intraocular pressure. This is taken as evidence that the pressure regulating mechanism is becoming less able to control the changes in intraocular pressure caused by variations in aqueous outflow. Swanljung and Blodi,[27] Kronfeld[22] and de Roetth[8] have pointed out that in the glaucomatous eye the facility of outflow is relatively rigid, changing very little in response to provocative testing or other stimulus.

There is good evidence that the trabecular meshwork is a pressure regulating mechanism.[2,10,11,31] The degenerative changes seen in the trabeculae of eyes with open angle glaucoma can explain the failure of the pressure regulating mechanism in this disease.

There is a large familiar factor in the incidence of primary open angle glaucoma. It has been estimated that in 13 to 25 per cent of the cases of primary open angle glaucoma, some member of the patient's family has the disease.[8,21] Further studies show that in families with some member having the disease there is a higher percentage than usual of individuals with a "high normal" tension.[21]

Bennett[5] has found that the life expectancy of patients with glaucoma is normal. If early generalized sclerosis or degeneration of connective tissue (which has been suggested as a possible cause of primary open angle glaucoma) occurred in these people, we would expect that their life span would be shortened.

We have seen that some trabecular change occurs in normal eyes of some aged individuals. We know that as the age increases in normal individuals, the tension gradually rises, and this gradual increase in tension with age could conceivably be due to slight damage to the pressure regulating mechanism.

We know that prolonged increased intraocular pressure will damage the optic nerve, the ciliary body, and now, in cases of secondary glaucoma, the trabecular meshwork.

In primary open angle glaucoma, the individual must have vulnerable trabeculae, possibly inherited, which are injured by a level of pressure lower than that of other individuals and lower than that which would ordinarily damage the optic nerve or ciliary body. For example, if an individual had a trabecular meshwork which would be injured whenever the tension rose to 24 mm., and if at the age of 38 the tension had risen (with the normal decrease in facility of outflow with age) to the point where it reached 24 mm. daily at the peak of the diurnal curve, trabecular damage as described earlier would occur daily, causing the facility of outflow to

steadily decrease. The optic nerve might not begin to suffer until the tension was reaching 32 mm., which might be years later. The glaucoma had begun, however, when the tension was reaching 24 mm., a level at which progressive degeneration of the trabecular meshwork was assured.

It is also possible that the predisposed trabeculae, as they grow older, "wear out" like joints do in a patient with hypertrophic arthritis, so that they are no longer able to withstand the normal pressures that they formerly could, and are damaged by these normal pressures.

In brief, according to this theory, primary open angle glaucoma is caused by the presence of vulnerable trabeculae or a pressure regulating mechanism which is damaged by levels of intraocular pressure which ordinarily do not damage the trabeculae or optic nerve. This injury interferes with aqueous drainage and promotes an increase in intraocular pressure which further damages the trabecular meshwork.

CLINICAL IMPLICATIONS

This new concept of the pathogenesis of open angle glaucoma is attractive because with it one can readily explain many of the puzzling clinical findings of glaucoma.

a. *Juvenile glaucoma* can be thought of as occurring in a patient with very susceptible trabeculae so that the critical tension is very low and is reached at an early age, producing primary open angle glaucoma very early.

b. *Combined glaucoma.* Although it is undoubtedly true that some patients with combined glaucoma (patients with both a narrow angle glaucoma and a reduced facility of outflow when the angles are open) start out with primary open angle glaucoma and later develop an enlarged lens and a superimposed narrow angle mechanism, this new concept suggests that in some cases the reverse may be true. That is, in some cases of narrow angle glaucoma, the periodic elevations of tension may cause enough trabecular damage to cause a reduced facility of outflow even though no synechias are present and the angles are open.

c. *Low tension glaucoma* can be thought of as occurring in an individual in whom the optic nerve and retina are susceptible to damage by relatively low levels of intraocular pressure but the trabecular meshwork, the pressure regulating mechanism, is normally resistant and is not damaged excessively. Consequently, there is no progressive decrease in aqueous outflow even though the optic nerve undergoes degeneration.

d. *Secondary glaucoma.* As described earlier, cases of secondary glaucoma in which the original cause of the rise in tension has disappeared but the glaucoma remains or reappears even though no synechias are present can be explained in the same manner; namely, that trabecular damage occurred during the original period of elevated tension.

e. *Management of early primary open angle glaucoma.* In primary glaucoma, if pressure is what caused the progressive degeneration of the meshwork and the progressive decrease in facility of outflow, then early diagnosis and treatment is of the utmost importance. It is during the earliest period of the disease, when the pressure has not reached a level that we ordinarily think of as pathologic, and before optic nerve changes have occurred, when the greatest opportunity to halt the process of trabecular damage is presented. If our conception of open angle glaucoma is true, then our aim in treatment must be to lower the intraocular pressure *enough* to avoid further trabecular damage. This desired tension may be far below that necessary to prevent loss of field. To illustrate, a patient with early

open angle glaucoma may, with miotics, have his tension lowered to 28 mm. Hg. At this level, over a period of a year or two no progression of the field defect may occur. But if trabecular damage is occurring at this level of intraocular pressure, the pressure will eventually rise. If the tension could have been lowered sufficiently, perhaps to 22 mm., it is possible that no further trabecular damage would occur, and the glaucoma would never progress.

Recently Goedbloed[15] in a very interesting study has pointed out that the prognosis and response to treatment of primary open angle glaucoma is much better if the diagnosis is made very early before the appearance of any field defect, and Smillie and his colleagues[26] and Brav and Kirber[6] have made similar statements.

Fralick[12] has stated that families of patients with primary open angle glaucoma are a fertile field for glaucoma case finding and has suggested periodic glaucoma workups for the relatives of patients with the disease after the age of 40 years. If failure of the pressure regulating mechanism of these individuals becomes manifest, treatment with miotics should be begun early with, according to our new concept, good expectations for success in treatment.

Becker and Christensen[3] and Hildreth and Becker[19] have also recently stressed the clinical importance of early determination of primary open angle glaucoma and have delineated new techniques of early case findings.

Confirmation

The pathologic changes in cases of primary and secondary glaucoma can be readily seen. The theory of the pathogenesis of primary open angle glaucoma, however, must await final proof. We are at present attempting to see whether prolonged elevation of intraocular pressure in animals will produce these degenerative lesions of the meshwork. We must make further careful histologic studies of the filtration angles of cases of primary and secondary glaucoma. Finally, the clinical implications of the theory must be tested in the crucible of clinical experience.

SUMMARY

Histologic study of the trabecular meshwork of 16 cases of primary open angle glaucoma and 11 cases of secondary glaucoma with open filtration angles in meridional, transverse and tangential sections reveal marked degenerative changes in the meshwork and Schlemm's canal, beginning in the external portion of the trabecular meshwork. The location and character of the degenerative changes indicate that they are responsible for the decreased facility of aqueous outflow characteristic of primary open angle glaucoma. It is apparent from the congruity of the histologic alterations in primary glaucoma and in secondary glaucoma with open filtration angles that these degenerative changes are caused by prolonged increased intraocular pressure.

A new theory of the pathogenesis of primary open angle glaucoma is proposed. It is suggested that persons destined to have primary open angle glaucoma have relatively vulnerable trabeculae which are injured by levels of intraocular pressure which ordinarily do not damage the trabeculae or optic nerve. When the curve of intraocular pressure, which normally slowly rises with advancing age, reaches the point where enough trabecular degeneration has occurred to cause insufficiency of the pressure regulating mechanism, progressive glaucoma is assured unless treatment is instituted. This theory implies that the aim of therapy in early glaucoma should be to lower the intraocular pressure sufficiently to prevent further trabecular

damage, rather than lowering it enough merely to prevent damage to the optic nerve; for if the trabeculae continue to be injured, the pressure will inevitably rise.

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DISCUSSION

HERBERT M. KATZIN, M.D., New York: I am very happy to see that Dr. Flocks has confirmed the finding of obstructive changes in the region of the canal of Schlemm. This type of serial section study is extremely time consuming; I know how much work it represents, but I feel that it has been worth while. In the Eye Bank our interest in this subject began with anatomic reconstructions by Dr. Teng in 1953; he is still at it.

Surely the prolonged high pressure mentioned by Dr. Flocks may itself produce changes in the angle of the anterior chamber. I would like to offer another concept for your consideration: We have observed that when the intraocular fluids gain access to the collagenous structures of the eye, degeneration ensues unless the collagen is protected by a continuous layer of endothelium. Our observations were first made on Eye Bank specimens. Apparently some substance in the aqueous causes the collagen fibers to swell, to lose their ground substance and supporting framework, and to disperse. The usual staining affinity is lost, and finally there are only fine irregular fibers and a flocculent, amorphous substance left. The effect is not directly on the cells, but on the supporting structures. These changes were observed in the angle of the anterior chamber, in the region of new trephine wounds, and, experimentally, in pieces of sclera embedded in the anterior chamber.

Dr. Chi embedded pieces of sclera from the opposite eye into the anterior chamber of rabbits. In many instances, the implant became encapsulated by endothelial cells and remained unchanged for as long as thirteen months. In those instances in which the implant was not so encapsulated, collagenous degeneration occurred, similar to that seen in wide angle glaucoma.

GEORGIANA DVORAK-THEOBALD, M.D., Chicago: Dr. Flocks has presented his concept of the pathogenesis of open angle glaucoma.

Until we have the final answers on the subject of chronic open angle glaucoma there is much room for theorizing and speculation. If some of my remarks seem to contradict the author's concept, they are made only for the purpose of keeping the doors open. I do not believe that he, nor I, nor anyone can yet point out the primary cause of chronic open angle glaucoma. For years I have observed and studied many of the changes found in old glaucomatous eyes which are so beautifully shown in Dr. Flocks presentation. No one is more aware than I of the difficulty—and, at times, the impossibility—of determining which changes came first, or which caused which, when examining the old glaucomatous eyes after they finally reach the laboratory.

The author's concept, like that of many earlier workers, assumes that glaucoma patients possess a predisposition to premature or abnormal degeneration of the collagen and connective tissues of the trabeculae and canal of Schlemm regions. Dr. Flocks believes that early in the disease this degeneration is accelerated by abnormal fluctuations in the diurnal pressure, and that this in turn is altered by the premature degeneration. The slowly rising pressure with increasing age presumably aggravates the process. He interprets his observations on eyes with advanced open angle glaucoma and eyes with secondary glaucoma as degeneration and fibrosis of the tissues adjacent to Schlemm's canal; areas of closure and adhesions within the canal with some recanalization; thickening of the trabeculae; partial closure of trabecular pores, and a variable new vessel formation on the medial side of the canal.

I can see how one might misinterpret some of the changes seen in tangential sections, and I would like to show some of my slides on the normal anatomy of this area to demonstrate how multiple channels, adhesions and the presence of certain blood vessels might be misinterpreted as pathologic.

In the eyes of the patient who died of multiple myeloma, which were the only early glaucomatous eyes in the author's series, I was not very impressed with the lesions described and illustrated. I do not see how one can draw serious conclusions with regard to

such changes without a study of many more early glaucomatous eyes in all age groups.

Dr. Flocks' theory of the early changes in the trabecular area, and the causation thereof, is not essentially new. He correlates the clinical and pathologic findings well, but I do not believe the evidence presented is adequate to prove his theory.

Dr. Flocks states that the obstruction could not be beyond the canal of Schlemm because if it were, the canal would be dilated and the trabecular spaces open. In our paper on this subject, we proposed that the connective tissue changes in such eyes not only involved the sclera, but the trabeculae as well, and that the canal of Schlemm and its afferent and efferent channels are "caught in a vice," so to speak, resulting in embarrassment to outflow. We admit some of our observations were speculative, even though based on direct observation, and are not able to say which comes first, the glaucoma or the changes in the connective tissue. Dr. Flocks is being even more speculative in assuming from the material presented an inherent weakness in the trabeculae, which can be damaged by a so-called normal ocular tension and start the vicious cycle.

I cannot see how one could derive a valid conclusion as to the etiologic basis of open angle glaucoma by comparison of the changes in eyes with old chronic glaucoma with those in previously inflamed eyes with secondary glaucoma.

We know that the pathogenesis is different in these two types of glaucoma. Therefore, I believe that most of the trabecular changes shown are the result of prolonged elevation of ocular tension, regardless of the cause of the elevation in pressure.

I would suggest that further studies be made along the following lines before further presentations are made on this subject:

1. Is there a substance in the trabecular spaces which changes with age and renders the meshwork less permeable?
2. What are the normal changes in the trabeculae, the intratrabecular spaces, Schlemm's canal, sclera and collector channels with increasing age?
3. What is the tissue comprising the trabecular fibers, and how does it react to injury?

Until these and subsequent problems are answered, we are hypothesizing.

DR. FLOCKS: I wish to thank Dr. Katzin and Dr. Theobald for their discussions.

Dr. Katzin and I are essentially in agreement as to the presence of degeneration in the trabecular meshwork and its site in the meshwork in primary glaucoma. In these respects, this paper confirms the work of his group. Dr. Katzin's speculation as to the cause of the degeneration is very interesting, but my finding that the same changes occur in the trabecular meshwork of eyes with secondary glaucoma due to many different causes would tend not to support his theory.

I am happy to see that Dr. Theobald, since her last paper on the pathology of the meshwork of glaucoma, has changed her viewpoint. In that paper she speculated that the cause of the obstruction was in the sclera and that it was due to an aging process, with changes in the connective tissue. But now she suggests that further study be directed toward Sondermann's canals in the external trabecular meshwork or the inner wall of Schlemm's canal, which is the same region in which I have demonstrated pathologic changes.

I did not mention Sondermann's canals in this paper, because I have previously discussed them in a study made several years ago, of the normal anatomy of the trabecular meshwork, particularly as seen in tangential sections. The structures known as Sondermann's canals have been noticed by a number of workers, and I have seen them. I do not believe they are true passageways into Schlemm's canal because they are too large.

Considerable experimental work has recently been done to measure the pore size of the meshwork by putting various substances of different sizes into the anterior chamber and seeing which kind and size will filter through. Francois, using a radiographic technique, and Huggert, using chemicals and bacteria of different sizes, have shown that the critical openings between anterior chamber and Schlemm's canal are two to three microns in size. Sondermann's canals are much larger. The one that Dr. Theobald gave as a classic example appears to be four times the size of a red cell—at least 30 microns in size.

When studying the trabecular meshwork in serial tangential section, one can measure the size of the openings. I have noted that progressing from the anterior chamber side, the elliptical corneoscleral openings are quite large, usually about 10 to 20 microns in their smallest diameter. The openings appear slightly smaller as Schlemm's canal is approached, until just beneath the inner wall of Schlemm's canal, no openings are seen, merely a granular, pale, more or less homogeneous tissue of varying thickness. In this tissue the smallest openings must be present. This is where the pigment collects, and this is where the pathologic changes are seen. So I do not believe

that Sondermann's canals are the true openings of the trabecular meshwork into Schlemm's canal; I believe that they are merely outpouchings of Schlemm's canal into the trabecular meshwork and that they have a lining on them.

I certainly agree with Dr. Theobald that we should keep this discussion open. This theory is not proposed as more than a theory. Concerning the pathologic changes, I don't agree. I know that I can tell the difference between the trabecular meshwork of these glaucomatous eyes and that of normal eyes; and that I cannot tell the difference between the trabecular meshwork of the eyes with secondary glaucoma and that of those with primary glaucoma. So my conclusion must be that the pathologic changes are caused by the same thing, whether the glaucoma is of primary or of secondary origin.

Dr. Theobald speaks of these as old glaucomatous eyes. Actually, every one of these eyes is a very rare specimen, because we seldom see enucleated eyes without peripheral anterior synechias in cases of glaucoma. By the time they are enucleated, they almost always have synechias. Twelve of these eyes with primary glaucoma came from the Armed Forces Institute of Pathology. I culled them from their 40,000 specimens or so, and they were all that I could find. Two of the eyes came from the Stanford Eye Pathology Laboratory. That was all we had in our laboratory. Two came from the Stanford Eye Bank. These may be old glaucomatous eyes, but very few such eyes of primary glaucoma without synechias have been reported. To my knowledge, this is the largest series of such cases that has ever been studied.

Again, I most assuredly am not proposing this as the final word or theory as to the cause of glaucoma. I have merely described pathologic changes in the trabecular meshwork and presented a hypothesis suggested by the facts.

ALLERGIC HEADACHES

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THE debatable role of allergy in the causation of certain headache syndromes has been the subject of argument, pro and con, since Vaughan's original article[33] in 1927. Since then, many other authors in this country including Rowe,[26] Eyermann, Rinkel,[23,24] Randolph,[21] Unger,[32] Ogden,[17] and others have repeatedly emphasized the role of allergy as an origin of headache. In reviewing more recent articles on this subject of head pain, it appears, however, that only a few cases in which allergic factors are involved are specifically diagnosed and treated as such. This is indeed discouraging to those physicians who have demonstrated the cause and effect relationship of food and inhalants to the production of head pain.[2,8,25(g)] The apparent reason for this lack of association seems to stem from the fact that, in general, the results of management of allergic headaches, in the past, have been poor. This has been true even though it has been recognized for years that a specific hypersensitivity could produce headache. This cephalalgia was classified at first as migraine. As a matter of fact, the early writers used the terms headache and migraine synonymously. Eyermann,[4] in 1931, was the first to note that there were many headaches that did not fit into the classical migraine pattern and suggested the term *allergic headache* rather than *allergic migraine*. He noted that many times the patient had nasal symptoms, mucoid discharge and a generalized distribution of head pain—frontal, diffuse or bilateral rather than hemicranial. He found several patients with positive skin reactions to inhalants, and a high incidence of food sensitivity. He also noted concomitant reactions to food and inhalants. Rinkel and Balyeat[24] confirmed these findings in 1932. Goltman[6] at this time also reported good results from treating headache by hyposensitization of his patients to the offending inhalant, e.g., orris root, pollens, or danders. Forman[5] and Sheldon and Randolph[29] reported similar series of cases. Since 1935 the literature has been rather barren on the subject until the recent past.

Why has it been that the allergic origin of head pain has not been recognized in the diagnosis with greater frequency? The answer is threefold:

1. Because the affecting allergen, whether it be food or inhalant has not been investigated completely
2. Because of the inherent difficulties in the specific diagnosis of food allergy
3. Because we persist in thinking of headaches in terms of their description or classification rather than in terms of their etiologic basis.

Elaborating on the first cause, it is the consensus among allergists today that food plays a much greater part in the production of head pain than inhalants do. Unfortunately, the diagnosis and treatment of food allergy has not kept step with inhalant allergy, which today has almost reached perfection in its management. It must not be forgotten

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that concomitant sensitization to foods and inhalants is possible in the same individual. Vaughan,[34] Ogden[16] and Rinkel[25(g)] have reported on this subject.

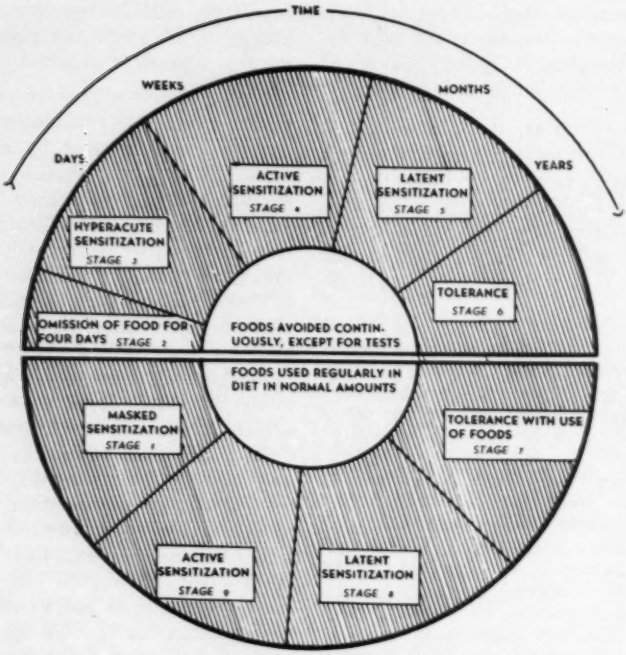
In elaborating on the second reason for our failure to recognize the allergic factor in head pain, we feel that the nature of food allergy has not been completely investigated or understood. Rinkel and his colleagues aptly express this in their recent book on food allergy.[25(a)]

1. There has been an unjustified faith in the reliability of skin test procedures for diagnosis. These techniques, often being mechanically performed and literally interpreted, have retarded the development of this subject.
2. There has been a wide-spread lack of knowledge concerning the dynamic nature of the food sensitization process.
3. The problem is complicated by the time and the amount of work required to make the specific diagnosis.
4. Our lack of complete knowledge concerning the sources of certain major foods in the diet, such as corn, soy bean or cotton seed, for instance, has often been responsible for the failure to effect complete relief in certain cases even though they were correctly diagnosed.
5. There has been difficulty on the part of both physicians and patients to place existing knowledge into actual practice. As in the past, the ease to disbelieve a new concept frequently overcomes scientific curiosity and its attendant desire to explore a new field.

At this time it seems important to define food allergy. This term refers to definite clinical manifestations produced by foods under correct conditions of testing; these reactions must exhibit specificity and must occur constantly. In other words, the test must demonstrate a cause and effect relationship between the ingestion of a specific food and the production or accentuation of allergic symptoms. An allergen food, therefore, is one that produces headache, induces asthma, increases hay fever or gives rise to any constant symptom pattern that repeatedly exhibits specificity.[25(b)] Food allergy is therefore not necessarily related to a positive skin test.

The dynamic nature of food allergy has been completely misunderstood, misinterpreted or ignored by most physicians. This is unfortunate, because a knowledge of its mechanism and the factors which modify the clinical manifestation is essential to the establishment of the specific diagnosis. Rinkel[25(d)] classifies food allergy into (1) *cyclic* or intermittent sensitization and (2) *fixed* or constant sensitization. In the former, the incidence of usage of a specific food in the diet tends to increase the degree of sensitivity, and its omission tends to develop tolerance (tolerance here being only relative, not absolute). In the latter—*fixed* food sensitization, the degree of sensitivity is not affected by the incidence of the allergenic food in the diet. Thus a patient may be just as allergic two years hence as he is today, without having eaten the food in the meantime. Cyclic sensitization, therefore, tends to be dynamic and fixed sensitization, static; however, many times there is no sharp difference between the two except in the classical cases.

The successive states in the degree of sensitization in cyclic food allergy may be represented graphically, reading clockwise (fig. 1). The changes occurring with the omission of the food are found in the upper half of the chart, those following the reintroduction of the food are shown in the lower half. One begins with *masked* sensitization, because this is the most important clinical feature of this type of food allergy and it is during this stage that most patients seek medical advice. The brief (4 day) omission of a certain food leads to the development of a hyperacute stage. With omission of this food over a longer period, the stages of active and latent sensitization and, finally, tolerance develop. When the allergenic food is reintro-



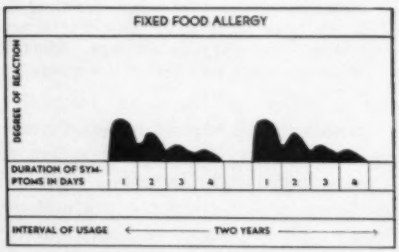
THE CHANGES IN THE DEGREE OF SENSITIZATION IN CYCLIC FOOD ALLERGY

FIGURE 1.[20(c)]

duced into the diet, the same stages develop in reverse order; if the food is continued at intervals of less than 72 hours, masking again occurs. Rinkel has defined this phenomenon as cyclic food allergy because it completes a cycle from masking to tolerance to masking.

Rinkel's impression of fixed food allergy is shown in figure 2. Here the patient's reaction to the allergenic food after its absence from his diet for two years is the same as it was during the initial test. (The difference between fixed and cyclic food allergy lies in the *degree* of sensitization only.)

The patient with head pain due to a specific food allergy has different reactions to the specific food depending upon the phase or stage of his sensitization. If his sensitization is in the masked stage, there is a decrease in or absence of his symptoms following ingestion of a food to which he is specifically sensi-



THE NATURE OF THE REACTION IN FIXED FOOD ALLERGY

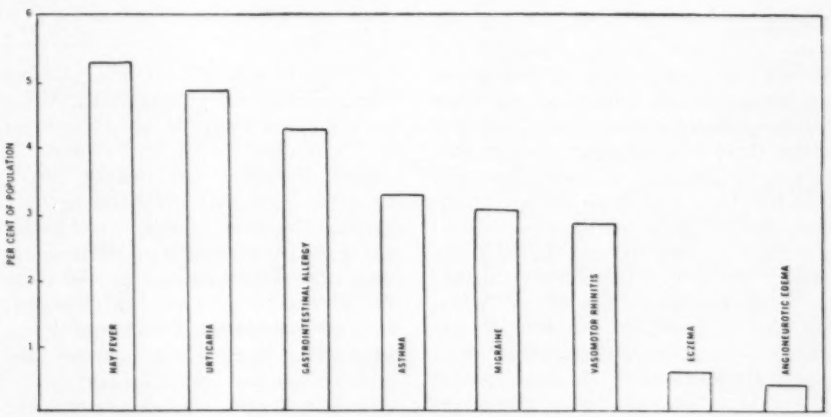
FIGURE 2.[21]

tive. If it is in the hyperactive or acute stage, there is an immediate increase in his symptoms following the ingestion of the food. If he is undergoing the tolerant phase, there is no reaction. This peculiar facet of food allergy has probably been responsible for more undiagnosed cases of headache than any other factor. Now, if a previously masked food allergen is returned to the diet after a period of avoidance from 96 to 120 hours, the patient is immediately thrown into an acute reaction, resulting in headache. If this specific food is eliminated for several months and then reintroduced, the patient may experience a slight headache, or possibly only a feeling of fatigue (active sensitization). The patient lapses gradually into a latent sensitization as avoidance of the allergen food continues. Readmission of the food for the first time fails to cause a headache, but on the next exposure the food causes a typical reaction. It is difficult for the neophyte to comprehend this phase of the allergic reaction because ingestion of the allergenic food produces no symptoms of sensitivity, but the next time the food is eaten a typical reaction of headache is precipitated. The final stage is tolerance, which is said to exist when no reaction follows ingestion of the specific food. Complete tolerance to an allergenic food is rarely, if ever, attained.

If allergy is a factor in headache, foods are by far the predominant cause, inhalants playing a poor second. Of the inhalants, house dust, molds, and the pollens are the chief sources of trouble. As stated before, many patients who have a food allergy become worse, experiencing flares in their food allergy when some new inhalant is introduced into the atmosphere. This is especially true when short ragweed appears. Carpenter,[2] who has written on headache caused by inhalant allergies, concludes:

> Diagnosis of inhalant allergic headache is made by (1) a never-ending suspicion of allergic etiology behind every functional headache; (2) a detailed and accurate history; (3) identification of the allergen by history and skin tests; (4) relief of symptoms conse-

COMPARATIVE INCIDENCE OF MAJOR ALLERGIC DISORDERS.*



(From a study of an entire community of 508 persons reported by Vaughan and Black[3])
*Some had two or more types of major allergy

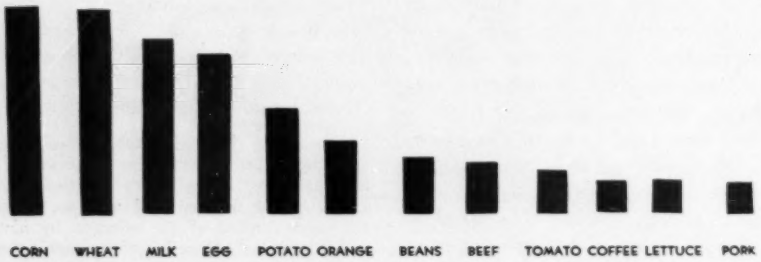
FIGURE 3.[26]

quent to specific avoidance and/or hyposensitization [as advocated by Hansel[7]].

Vaughan and Black[35] have made a study of the incidence of allergic headache (migraine) in the cross section of a typical village. They found that of all the allergic symptoms, migraine (headaches) ranks fifth in order of importance in allergic manifestations (fig. 3).

HISTORY

Shuey[30] states that one would experience great difficulty in immediately classifying any given headache as an allergic cephalalgia solely on the basis of the history that the patient recites. Since there is usually nothing typical of such cases, there are not characteristic pain patterns.[2] The type of onset, however, may provide a clue to an allergic causation. Hansel[7] feels that the onset is usually gradual, since the mechanism of the allergic headache is based not altogether on a vasoconstrictor-vasodilator action but also on capillary permeability and edema. Several other characteristic factors may direct one's attention toward allergy as an etiologic factor. These are (1) a personal history of allergic disturbances such as eczema, angioedema, urticaria, allergic rhinitis; (2) a strong family history of allergies; and (3) concomitant allergies such as seasonal and perennial rhinitis, and urticaria.[5] With the help of history outlines, as advocated by Anderson and Rubin of New Orleans, no facet of the history is overlooked. A complete history is very helpful and almost diagnostic (with the help of skin tests) in inhalant allergy. While valuable information may be obtained from the history in relation to food allergens eaten at infrequent intervals, because of masked food reactions the history cannot be relied upon for detection of many specific food sensitizations. One of Rinkel's patients suffering from a masked allergy was asked, "What foods do you know to be a cause of your symptoms?"[25(e)] She replied, "I suspect pineapple and tomato, but I know for sure that corn and egg are agreeable because I always feel better after eating them and I eat them nearly every day." Corn and egg proved to be her only two important food allergies; pineapple and tomato did not cause symptoms. Figure 4 presents the incidence of common food allergens.



INCIDENCE OF COMMON ALLERGENS.

FIGURE 4.[30]

Thus we see that a good history, while important, may mislead one in relation to food sensitivities. Probably the one point most important in this respect is

the quantitative food intake history—the individual eating habits of the patient. We must also elicit information regarding the influence of previous dietary manipulation, the evaluation of childhood dietary factors, food dislikes and food suspects, and the age at which certain foods were disliked or suspected as being allergenic.

SYMPTOMATOLOGY

1. In the symptomatology of allergic headaches most authors[8,23,32] agree that there must be a family history of chronic headaches present.
2. There is also a significantly higher familial history of allergy present.[18]
3. The symptoms vary with each attack. (There is no select set of symptoms present.)
4. No organic cause can be found to explain the headache.
5. The etiologic basis of the headache may be food or inhalants. The allergic factor in headaches is mainly one of food.
6. The patient's symptoms must include the criteria for food allergy:
   a. Itching of the nose or the roof of the mouth must be present.
   b. The patient must awaken out of a sound sleep in the middle of the night with an acute exacerbation of symptoms.
   c. There should be a history of exacerbations lasting from 18 to 72 hours.
7. The importance of chronic fatigue should be studied with great care. Fatigue usually precedes the headache. Several concomitant allergies may exhaust the patient.
8. Increase in the pulse (tachycardia) is present in many cases following the ingestion of allergen food. When this is present it can indicate a delayed reaction as well as a positive one.[3]
9. Pain in the posterior cervical muscles (nuchal pain) associated with occipital headache under stress is one of the cardinal symptoms of a food allergy.[27]
10. Leg ache, especially at night, is present in many cases.[25(g)]
11. The age of the patient at onset of headaches may be no more than 5 years.
12. The frequency of headaches is variable: every one or two months, every week, every weekend, or every day.
13. The patient may complain of varying degrees of weakness and states many times that he is as tired in the morning as he was at bedtime. He especially feels worse in the morning and may be improved after breakfast. He is much better in the evening and frequently does not do his important work until late afternoon or evening.
14. Irritability may be present and may be associated with various degrees of depression, even to the point of mental confusion. This is especially true in children.
15. There may or may not be cortical symptoms, depending on the shock organ involved.
16. The location of the headache has little to do with the ability to relieve the symptoms.
17. The type of pain is not important in diagnosis except in helping to classify the headache.
18. Pallor is present in many cases of headache. This is not a symptom of headache *per se* but one of allergy in general.
19. Insomnia is present in varying degrees.
20. The terminal phase of the attack is characterized by polyuria, with resultant loss of edematous weight.[8]

PROGNOSIS

1. The prognosis is good irrespective of the age at onset.
2. The duration of the headache (in years) has little to do with the final result.

DIAGNOSIS

An accurate and detailed history of the patient is quite essential in establishing allergy as a cause of cephalalgia. In inhalant allergy, the skin tests are diagnostic, and head pain will be relieved by proper dosage or aggravated by too large a dose. In food allergy, however, we do *not* make the diagnosis by elimination of the offending allergen but by its readmission to the diet. When this is done under proper conditions of testing and the reactions are constant and exhibit specificity, the patient with acute allergy will have an experience of head pain within ten to thirty minutes postprandially, and a marked leucopenia of at least 10 per cent.

The diagnostic approach to food allergy consists of three integrated procedures: (1) the use of individual food tests to determine the specific effect of certain foods (as stated above); (2) the use of a basic diet consisting only of those foods to which the patient is found compatible; and (3) the deliberate addition of foods to this basic diet one at a time.

CLASSIFICATION

We have stated before that one would experience great difficulty in classifying any given headache as an allergic cephalalgia because of the absence of characteristic pain patterns and the lack of a typical symptom complex. Yet we feel, as does Unger[31] that allergic headaches tend to fall into three main groups. This is due to the fact that the shock organ involved varies in different patients and in different attacks. These three groups are: (1) frank nasal allergy; (2) "allergic headaches" secondary to edema of the brain and/or meninges (affecting the smaller vessels); and (3) vascular headaches, including migraine, histaminic cephalalgia, myalgia, and frontal headaches (affecting the greater vessels).

1. *Frank Nasal Allergy*

This most common type of nasal allergy causes edema of the nasal passages and sometimes gives rise to a headache that has been labeled "sinus headache." This type of head pain is thought to be secondary to pressure on pain sensitive turbinates, ostia and ducts of the sinuses. That this is the mechanism of "sinus headaches" has been proved by Holmes, Goodell, Wolf and Wolff.[12] They also demonstrated that in an edematous nose, nerve stimuli, such as cold air and misdirected air currents, can cause typical sinus pain across the bridge of the nose, over the zygoma and over and behind the eyes. These writers demonstrated that the sinuses themselves are relatively insensitive to pain but that allergic or infectious edema of the ostial membranes can cause severe headaches. Many patients suffering from nasal allergy say that their headaches are due solely to nasal blockage and that relief of the edema brings about relief of the headache. Many other patients with allergic rhinitis, completely unable to breathe through the nose, do not complain of headache.

Of course, in this type of headache, it is felt that the "shock organ" is located mainly in the nose.

2. *Allergic Headaches*

This type of headache is the "garden variety" allergic cephalalgia that most physicians easily recognize as allergic head pain. It may be frontal, occipital, parietal or generalized. Ogden[2c] reports that in a survey of 4,634 patients with headaches, 47.1 per cent of the total pop-

ulation, or 72.7 per cent of the headache fraction complain of frontal headache; 10.1 per cent of the headaches were occipital. Many of the frontal headaches were of allergic origin and could be reproduced at will by giving overdoses of antigen. Interestingly enough, many of the patients with frontal headache had completely open noses.

Ogden[19] feels that these frontal headaches are mainly vascular in nature (fig. 5). The anterior and posterior ethmoidal arteries arise from the ophthalmic artery as it runs forward along the medial wall of the orbit. The posterior ethmoidal artery supplies the posterior and upper part of the lateral wall of the nasal cavity. The anterior ethmoidal artery passes through the anterior ethmoidal canal with the anterior ethmoidal nerve, enters the anterior fossa of the skull, and crosses the cribriform plate of the ethmoid, passes through an aperture at the lateral side of the nasal slit into the anterior nasal cavity, where it descends to the tip of the nose. It gives off meningeal branches in the anterior cranial fossa, and supplies the anterior and middle ethmoid sinuses, the frontal sinuses, the nasal mucoperiosteum and the skin of the dorsum of the nose. Thus, Ogden reasons that autonomic stimulation in the nose involving the anterior or posterior ethmoidal arteries could very easily stimulate vasoconstriction-vasodilation phenomena in the anterior meningeal arteries, resulting in pain. While this is fundamentally a vascular phenomenon, we must consider the nose and the affected vascular tree to be concomitant shock organs.

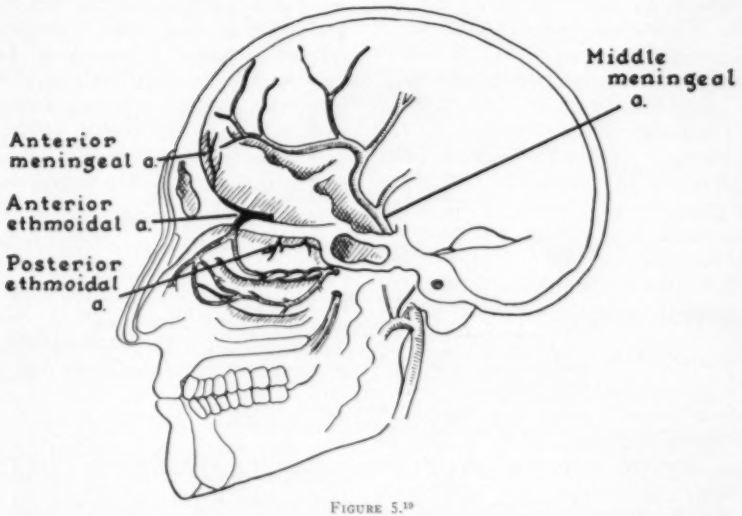


FIGURE 5.[19]

Eyermann[4] described this identical syndrome but believed it to be localized cerebral edema or hyperemia. Unger[31] feels that until either of the above theories is proved correct, localized mucosal edema at the sinus ostia is the logical mechanism to explain the cause of frontal headache.

Hansel[7] has described an allergic headache syndrome associated with cerebral and peripheral edema. Poly-

dypsia, peripheral edema especially of the hands, feet and face, and weight gain precede the attack, while polyuria, weight loss, and a feeling of well-being herald its end. This headache is truly generalized, with a bursting sense of pressure, dizziness, and various stages of mental confusion. It occurs in allergic individuals and is frequently accompanied by other allergic phenomena such as gastrointestinal dysfunction, urticaria, angioedema, atopic dermatitis and rhinitis. The entire attack may last from twelve to seventy-two hours, and can be reproduced by ingestion of an allergen food. While this type of headache is fundamentally vascular, it is secondary to perivascular transudate and its resultant complications rather than to vasodilation-vasoconstriction phenomena. The first may be considered as a minute process while the second may be thought of as a gross process; however, the latter, if continued long enough, will have the same final result as the former. In these headaches the shock organ is in the various parts of the brain and meninges.

3. *Vascular Headaches*

Of all the vascular headaches, migraine is probably the best known. Ogden[20] finds migraine present in 3.3 to 8.6 per cent of the cross section of the population, depending upon whether it was rigidly defined or broadly defined. (Vaughan[35] reported about 3.2 per cent.) Much has been written concerning the association of migraine and allergy. As late as 1952 two directly opposed articles appeared in the same issue of the *Journal of Allergy*, one by Unger and Unger[32] proving the association of allergy and migraine, and the other by Schwartz[28] disproving it. In commenting on this, Kallos and Kallos-Deffner[13] states:

> If you compare these and many other papers by advocates of the allergic or non-allergic nature of migraine, you will find that the conclusions are mostly built up on the same kind of evidence, such as case and family histories, the results of skin tests, and feeding experiments and the evaluation of the efficacy of anti-allergic treatment by specific desensitization and elimination diets. A number of clinicians are able to group and arrange this evidence in quite a plausible pattern, showing a co-relationship between migraine and allergy; in the hands of others, however, the opposite pattern will emerge. It seems to us quite probable that the firm *pre*formed belief of these investigators has really a great deal to do with the conclusions they finally reach.

Allergy has apparently been a factor in the causation of histaminic cephalalgia. MacNeal[15] reports successful treatment with rigid elimination diet, further stating that histamine desensitization was of no value. I have seen one case like this. The patient had typical symptoms of histaminic cephalalgia on exposure to onions and highly spiced food only when grass pollen was in the air—and he was not sensitive to grass!

Randolph[22] observed myalgia of the cervical muscles repeatedly following experimental ingestion of foods or exposure to inhalants in specifically sensitized persons. These reactions could be repeated at will and were relieved by avoidance of incriminated allergens, proving the basis of the thesis that these manifestatons were of allergic origin. This fact has been duplicated by many allergists and some feel that it is a cardinal symptom of food allergy.

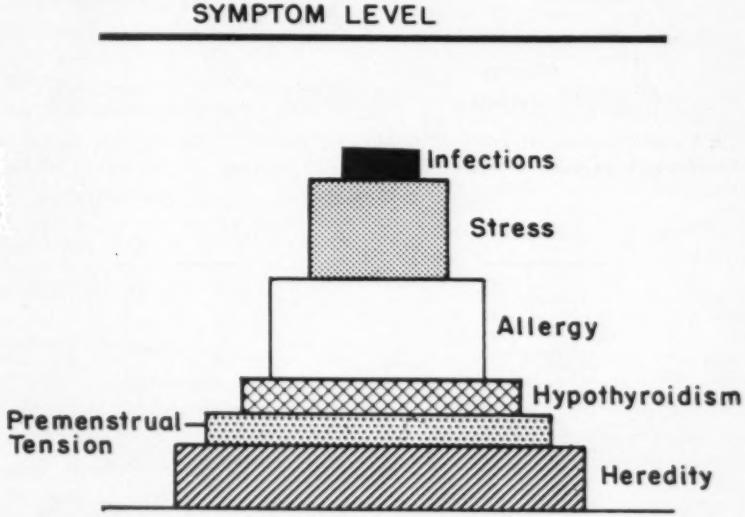
An interesting sideline on this facet of headache was reported by Braff.[1] One hundred twenty-four (124) cases of headache were treated by head traction, manipulation of the neck, intramuscular injection of 200 mg. of thiamin chloride three times a week for one or two months. A history of trauma to the neck was elicited in 49 per cent of the cases. He reports that 75 to 100 per cent improvement was obtained in 75 per cent of the cases. Patients with a history of headache due to specific foods could tolerate these foods after therapy, "suggesting that apparent food allergy

was secondary probably to vagal irritation induced reflexly by stimulation of the upper cervical nerves."

We all know that an accurate diagnosis of the allergic state is a very delicate task. Even in true allergic individuals not all co-existing disease syndromes need be of allergic origin. If we can show that some allergenic substance can provoke a disease syndrome, it must be understood that the syndrome can be repeated by other factors. We must remember that

> . . . the quality of tissue response is not dependent on the kind of stimulus or the way of stimulation but only on the structure and physiological activity of the tissue. Muscles will always contract, glandular cells will produce secretion, the vascular wall will change its tone, and permeability, and so on. The allergic stimulus is by no means an exception.[13]

It must be remembered that allergic reactions, states of autonomic dysfunction and endocrine imbalance can produce identical clinical symptoms.[11] Hilger[9] describes excellent results in the therapy of vascular headaches by use of thyroid extract. Leyton[14] reports excellent results in treatment of vascular headaches with anterior-pituitary-like hormones. I have reported excellent results in vascular headaches with the use of Bellergal.[10] Figure 6 will show you what we are driving at. Each facet (i.e., heredity, premenstrual tension, hypothyroidism, allergy, psychogenic factors and infection) of the multiple factors of

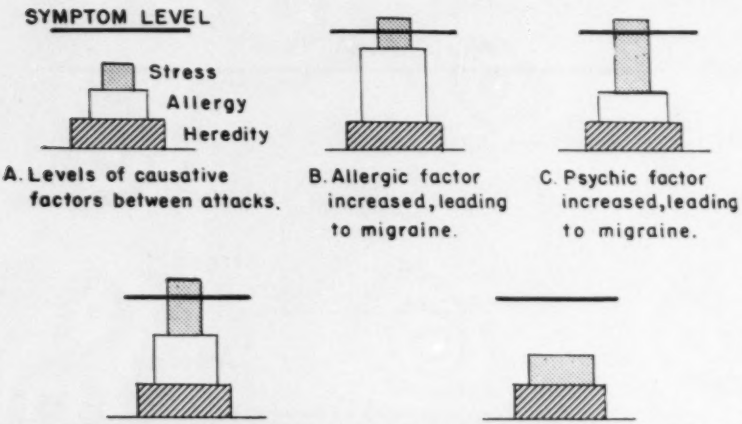


THE INTERRELATIONSHIP OF SOME OF THE MULTIPLE FACTORS IN THE ETIOLOGY OF MIGRAINE ATTACKS

FIGURE 6.

migraine (vascular headache) must be dissected free, analyzed and treated, to keep the patient below the symptom level. Figure 7, a simpler diagram, indicates the relationship of stress to allergy and heredity and the result of elimination of the allergic factor.[31]

seem to give the best results. I use Chlor-Trimeton, 0.1 cc. intramuscularly, or Sandostene and calcium, 10 cc. intravenously. If the drug is to be taken orally, I prescribe 8 to 12 mg. Chlor-Trimeton, 75 mg. Sandostene Spacetabs, or Dimetane Extentabs. Prophylactic measures should be aimed at isolating the offending allergen, and if it is a food, abstinence should be recommended. If it is an inhalant, proper low dosage injections should be administered. If the headache is vascular in nature or has vascular components, Cafergot may be employed to combat the acute attacks. When stress or autonomic imbalance is a factor, either in itself or associated with allergy, Bellergal Spacetabs or Compazine Spansules frequently aid in prolonging the quiescent period.


SYMPTOM LEVEL
Stress
Allergy
Heredity
A. Levels of causative factors between attacks.
B. Allergic factor increased, leading to migraine.
C. Psychic factor increased, leading to migraine.
D. Both allergic and psychic factors moderately increased, leading to migraine.
E. Allergic factor removed. Only extreme stress can produce attack.

RELATION OF ALLERGY AND STRESS IN CAUSE OF ATTACKS OF MIGRAINE

FIGURE 7.[31]

Before closing, I want to emphasize again the importance of fatigue and its close relationship to allergic headache. It frequently precedes headache and is always diagnostic of a subclinical headache syndrome. One must, also, always be aware of the inherent tolerance of the patient to any allergen; the degree of this tolerance greatly affects the prognosis.

TREATMENT

In the symptomatic treatment of allergic headache, the antihistaminic drugs

SUMMARY

1. Allergy is an important factor in headache.
2. The allergic factor in headache is principally one of food, inhalants playing a poor second.
3. Food allergy is dynamic in nature.
4. Fatigue frequently precedes allergic headache and is always diagnostic of a subclinical headache syndrome.
5. Allergic headaches are difficult to diagnose and, once diagnosed, difficult to treat.
6. The diagnosis and treatment of allergic headache can be aptly summed up in the following prayer attributed to Admiral Hart:

Dear God, give us strength to accept with serenity the things that cannot be changed. Give us courage to change the things that can and should be changed. And give us wisdom to distinguish one from the other.

———

Attributed to Admiral Hart, quoted by George Sessions Perry and Isabel Leighton in "Where Away" (Whittlesey)

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DISCUSSION

LAWRENCE R. BOIES, M.D., Minneapolis: This is a difficult paper to discuss—difficult because it deals in generalities. The author is not to blame; he has done a commendable job in "boiling down" into a few pages the "meat," so to speak, of the contributions to this subject of many workers, some of them renowned in the specialty of allergy.

In what I say, I too must generalize, because the author has made such statements as these:

"Because of the *inherent* difficulties in the *specific diagnosis of food allergy* headache has not been diagnosed with greater frequency."

The author defined an allergic food as follows:

"An allergenic food, therefore, is one that produces headache, induces asthma, increases hay fever (of course, he means any one or a combination of these disorders) or gives rise to any constant symptom pattern that repeatedly exhibits specificity."

But later in his discussion of symptomatology he states:

"The symptoms vary with each attack. (There is no select set of symptoms present.)"

"The patient must have the criteria of food allergy:

(a) Itching of the nose or roof of the mouth must be present
(b) The patient must awaken out of a sound sleep in the middle of the night with an acute exacerbation of symptoms
(c) There should be a history of exacerbations lasting 18 to 72 hours or more."

Rather widespread symptoms are listed by the author. Whenever such symptoms are so diverse in their location and type I have some doubts that these are all based on the same primary etiologic basis. I refer to such symptoms as chronic fatigue, irritability, tachycardia, insomnia, pallor, pain in the posterior cervical muscles associated with occipital headache under stress, leg ache (especially at night), a varying degree of weakness with or without early morning fatigue with a recovery of strength in late afternoon or evening, polyuria at the termination of the attack, etc.

I have some difficulty comprehending and accepting these divided states in the chart from Rinkel's textbook on the changes in the degree of sensitization in cyclic food allergy, and I am further confused in a reference to *cyclic* sensitization which tends to be dynamic, and *fixed* sensitization static when the author adds, "However, many times there is no sharp difference between the two except in classical cases." I wonder how many of the cases that are not "classical" have a correct interpretation of their cause.

Well, I could go on at considerable length, since I am generalizing.

I don't know just why I was asked to discuss this paper unless it was because I have expressed opinions previously regarding the symptom of head pain before Academy groups. In fact, my files show that I participated in instructional courses on this subject in 1940 and 1941 and for four consecutive years beginning in 1949. I know that I did not contribute anything that was new or suggest any magic panacea for the prevention or cure of head pain, but I do feel that I made some in my audiences feel that rhinology cannot be practiced through a nasal speculum alone and that we are expected to be physicians first and specialists secondarily.

I feel that Dr. Hilsinger's presentation makes a similar contribution and that he deserves our thanks for it.

ROBERT E. RYAN, M.D., St. Louis: The fact that allergic headache does exist cannot be denied; however, to state that such headaches as migraine, histaminic cephalalgia and myalgia are of allergic cause is, in my opinion, totally incorrect.

Within the past several years there has been a considerable amount of discussion by allergists concerning the etiology of migraine.

Eyermann in 1931 described a group of recurrent allergic headaches. These, however, did not follow the recognized migraine pattern. In many of these cases nasal obstruction and discharge were present and the headache was often of the diffuse type and bilateral. In many of the cases described by Eyermann, the patient experienced headache in the frontal region, and the pain would often migrate from this area and spread out over the entire head. These patients were all skin-tested and were found to be sensitive to certain foods and also to certain pollens, different patients being sensitive to different things. These headaches were called allergic headaches, not migraine, by Eyermann.

The symptomatology of migraine and allergic headache certainly cannot be called identical. Scotomas, photophobia and the gastrointestinal disturbances seen in migraine, causing the patient to state that he has a "sick headache," are not part of the picture of a typical case of allergic headache.

While MacNeal might report successful treatment of a case of histaminic cephalalgia, I am sure that Horton, who initially described this condition in 1936, is not of the opinion that it has an allergic basis. I have never seen a case of histaminic cephalalgia with an allergic background. Allergic headache responds well to antihistaminic therapy. This certainly is not true in histaminic cephalalgia.

Myalgia of the head is generally believed to be due to a physical allergy and not to an allergy of the antigen-antibody type. This has frequently been described in the literature by Williams.

As I previously stated, allergic headache does exist. However, in reviewing the headache patients seen in my office during a one-month period (January) I found that only two patients out of a total of 153 headache patients actually had what was diagnosed as allergic headache. This is 1.3 per cent.

I believe that a careful history is an important factor in diagnosing any type of headache problem. This is also true in cases of allergic headache. If one believes the case to represent an allergic headache problem, careful skin testing should be performed. This will further aid in establishing a correct diagnosis.

As for the symptomatology, I find that in most cases of allergic headache there are usually some symptoms present which are referable to the nose. Usually, there is some nasal obstruction and congestion. A watery rhinorrhea is present, and sometimes sneezing is a part of the picture. Upon examination, the nasal mucosa is found to be of the pale type which is commonly seen in cases of allergic rhinitis, and the mucosa is hypertrophic and rather edematous. Normally, the mucous membrane of allergic rhinitis is of the bluish-gray type. This does not mean that all patients with nasal allergies also have allergic headaches, but the reverse is true, and most patients with allergic headaches do have some allergic symptoms which are referable to the nose.

Allergic headaches usually occur only when the patient comes into contact with the allergen causing the trouble, whether it be an inhalant or a food.

The allergic type of headache is bilateral and frequently more pronounced in the frontal area, the vertex, or the occipital area. The duration of the attacks may vary from less than an hour to several days.

As pointed out by Dr. Hilsinger, the best type of treatment seems to be use of the antihistaminic drugs, avoidance of all causative factors, whether they be items of food or inhalants, and desensitization, when possible. If satisfactory results are not obtained, the corticosteroids might be tried. However, I do not recommend this therapy except as a last resort.

DR. HILSINGER: I want to thank Dr. Boies and Dr. Ryan.

This was a very difficult paper to write, because there have been very few contributions from otolaryngologists.

I have been a victim of just such an attack as we are discussing today, and these attacks in the middle of the night are quite real to me. It was discovered that I was sensitive to wheat; with the elimination of this cereal from my diet, the nocturnal symptoms disappeared.

What I mean by specificity in headaches is that a headache is caused by ingestion of a specific food but this headache does not always have to have a certain set of symptoms. For instance, in allergic migraine, on one occasion the patient may complain of homonomous hemianopsia, scintillating scotomas and unilateral headache and on the next exposure to the same food, the patient may complain of unilateral headache, scintillating scotomas and diarrhea, while on a third exposure, the symptoms may again vary. The one common factor is the headache.

I did not mean to imply that this is a very common type of headache. It isn't. It represents a very small group. As to histaminic cephalalgia, I agree with Dr. Ryan. It might be good to use the term *histaminic cephalalgia-like* or *myalgia-like headache*.

BLEEDING FOLLOWING TONSIL AND ADENOID OPERATIONS

DONALD F. PROCTOR, M.D.

CARLETON C. DOUGLASS, M.D.

BALTIMORE, MARYLAND

SOME forty years ago Dr. S. J. Crowe reported a series of 1,000 tonsillectomies performed at The Johns Hopkins Hospital.[4] Twelve of these patients (1.2 per cent) bled postoperatively and resuturing of the tonsillar fossa was required. The purpose of this paper is to review a contemporary series of tonsil and adenoid operations performed with the same operative technique as that employed by Dr. Crowe.

All such operations carried out in connection with the authors' private practices between July 1941 and March 1957, a total of 1,650, have been included in the study.

With minor variations these patients were managed according to the following plan[29]:

Preoperative Care

The patient was admitted to the hospital on the day prior to the operation for history taking, physical examination, blood count and urinalysis. Usually no special study of bleeding or clotting characteristics was done.

No patient in this series was given vitamin K, Adrenosem, or any other drug designed to prevent bleeding.

Preanesthetic medication consisted of a barbiturate and a belladonna derivative.

Anesthesia and Surgery

Ether was the major anesthetic agent used for children; since late 1942, Pentothal Sodium has been the major agent used for most adults. The endotracheal tube was rarely employed in children; in recent years it has been used invariably in adults.

A Davis-Crowe mouth gag was used to ensure a patent airway and good visualization of the operative field. With the patient in the Trendelenburg position, the adenoids were removed first and a postnasal pack was left in place until the conclusion of the operation.

The tonsils were removed by blunt dissection, with the snare being used at the base of the tongue. Persistent bleeding points were clamped and sutured with plain 2-0 catgut. The coagulating current was employed in about fifty adults.

An average of 1.5 sutures per patient was required in children and about 2.1 sutures per patient in adults. Approximately 8 per cent of the patients required no sutures and as many as four sutures were needed in about 4 per cent.

Postoperative Care

The patient generally left the hospital the day following the operation, and vigorous exercise was forbidden for at least eight days. Aspirin was permitted for the relief of pain.

Patients were followed as long as possible, the average follow-up for each patient being eight months.

One thousand of the patients were analyzed in considerable detail. Sixty-seven per cent were children. About 3 per cent were over 39 years of age (table I). Slightly over half were males. Three-fourths of the operations were

From the Division of Laryngology and Otology, The Johns Hopkins University School of Medicine.

Presented at the Sixty-Second Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 13-18, 1957, Chicago.

TABLE I

AGE DISTRIBUTION OF PATIENTS

YEARS	%
1-4	29.8
5-9	34.2
10-14	8.4
15-19	4.3
20-29	14.5
30-39	6.1
40-60	2.7

done under ether anesthesia. Seven per cent of the operations were adenoidectomies and 9 per cent were secondary tonsillectomies.

Eighteen per cent of the operations were performed in June, 12 per cent in November, and 11 per cent in May. Only 4 per cent were performed in September, and 5 per cent each in August and February.

Thirty-nine per cent of the patients had suffered from recurring bouts of severe tonsillitis (1 per cent had had peritonsillar abscesses). Thirty-nine per cent suffered from recurring, severe, and prolonged upper respiratory infections. Seventeen per cent had recurring ear infections and another 10 per cent had a conductive hearing impairment. The other indications for operation were mouth breathing, cough, cervical adenitis, sinusitis, general debility, fever of unknown origin, and suspected focus of infection.

One hundred thirty-nine patients were afflicted with serious systemic disorders. Thirty-one had congenital cardiac malformations including tetralogy of Fallot, transposition of the great vessels, interauricular septal defect, and pulmonic stenosis. Fourteen patients had other forms of heart disease, 10 had nephritis, and 57 had asthma or bronchiectasis. Diagnostic bronchoscopy was performed at the time of operation on 32 patients.

The analysis of the incidence of postoperative bleeding was made according to the following definitions:

a. Slight bleeding which stopped spontaneously has been called *insignificant*.

b. Any bleeding, no matter how slight, which required special care for its control has been called *significant*.

c. If either a blood transfusion or resuturing of the tonsillar fossa was required, it has been called *significant and severe*.

There were no deaths.

Insignificant bleeding occurred in 32 patients (2 per cent), and these instances will not be discussed further in this paper (table II).

TABLE II

INCIDENCE OF BLEEDING FOR 1,650 TONSIL AND ADENOID OPERATIONS

NONE	1,602	
INSIG.		32
SIGNIF.		16

TABLE III

SOURCE AND SEVERITY OF BLEEDING RELATED TO AGE

	0-10	11-20	21-30	31-40	41-50	51-
RESUTURE	TT					
BOTH	TT					
TRANSFUSE	A		T			
OTHER SIGNIFICANT	TTT TTA		TT	A	T	
INSIGNIFICANT	TTAA AAXX XXXX XXXX	TTXX	TTAX XXXX XX		X	A
AGE IN YEARS						

TABLE IV

SOURCE AND SEVERITY OF BLEEDING RELATED TO POSTOPERATIVE DAY

T-TONSIL
A-ADENOID
X-QUESTIONABLE

	1	2	3	4	5	6	7	8	9	10	11	12	13	14
RESUTURE	T				T									
BOTH	T				T									
TRANSFUSE	A						T							
OTHER SIGNIFICANT	TT	T			T		AA T	TT	T					
INSIGNIFICANT	TA XXX	XXX XXX T			XX	AAA ATX XX	XXX TA	T	X		X			TX
POST OP. DAY														

Sixteen patients had significant bleeding (0.97 per cent); 6 of these instances (0.36 per cent) were *significant and severe.*

In three patients (0.18 per cent) significant bleeding came from the nasopharynx, and in 13 (0.78 per cent), from the tonsillar fossa (table III).

Five patients (0.3 per cent) bled significantly during the first twenty-four postoperative hours, 4 (0.24 per cent) from the second through the fifth days, and 7 (0.43 per cent) from the seventh through the ninth days.

No statistically significant relationship could be discovered between bleeding and sex, age (table IV), anesthetic agent, indication for operation, ancillary systemic disease, or month of operation. No patient with a history of quinsy or with a serious systemic disorder bled significantly.

One of the authors had no patient with significant bleeding for a period of four years. He performed a consecutive series of over a thousand operations without a single patient requiring resuturing.

A proper evaluation of the literature on this subject is made difficult by the lack of definition of terms, especially the lack of a clear statement regarding severity of bleeding. The incidence of postoperative bleeding seems to vary between 1 and 20 per cent.[2,3,15,16,18-20,23,28] Approximately half of this bleeding occurs immediately after the operation and the remainder anytime up to two weeks later. Perhaps one-third of the bleeding is nasopharyngeal and the remainder occurs in the tonsillar fossa. Among 11 reports covering some 29,000 operations, there was an over-all incidence of 5 per cent postoperative bleeding (table V).

The best results seem to be those of Emerson,[6-8] and of Orzac.[24] Orzac has reported a series of 500 patients in whom antibiotics and Adrenosem were employed with no postoperative bleeding. To our minds this is too small a series to be of more than suggestive significance.

In a personal communication, Dr. Emerson has reported that among nearly 2,000 patients operated upon with his closed fossa technique, one suffered a severe hemorrhage and seven patients bled from the nasopharynx. This is by far the best series which has come to our attention. Whether his results are attributable to the closed fossa technique or to his own superior surgical ability can only be told when other surgeons report their experiences with this method.

TABLE V

INCIDENCE OF BLEEDING REPORTED IN THE LITERATURE

PATIENTS	BLEEDING
19,754 (3)*	1,216
1,000 (1)	12
2,000 (26)	8
1,744 (15)	55
200 (28)	17
443 (10)	29
233 (12)	16
916 (6)	54
1,808 (11)	95
800 (23)	9
385 (22)	8
29,283	1,519 — 5%

*References to bibliography.

As all of us know, some patients bleed to death. One report shows four deaths from hemorrhage among 20,000 tonsil and adenoid operations.[5]

Much of the recent literature has been devoted to studies of the effect of one drug or another on the reduction of the incidence of postoperative bleeding.[1,9-12,14,17,21,25-27]

One such study[13] reports an incidence of 14 per cent bleeding among 100 controls, 4 per cent bleeding when vitamins K and C were administered and no aspirin permitted, and 2 per cent when vitamins K and C and aspirin were given

to a second 100 patients. We would suggest that such a drop in the incidence of bleeding could be attributed to the employment of more meticulous surgical techniques.

Another author[22] investigating the value of vitamin K in counteracting the detrimental effect of aspirin showed an incidence of 1.4 per cent late secondary bleeding among 283 patients so protected as compared to an incidence of 10 per cent among those unprotected.

Since in our study of patients who received aspirin and no vitamin K, only 0.67 per cent of the patients bled after the first twenty-four hours, one would again question the significance of this finding.

In conclusion, among 1,650 operations upon the tonsils and adenoids, just under 1 per cent of the patients bled significantly postoperatively. It is our feeling that there is not as yet any adequate justification for the widely accepted belief that the use of vitamin K, Adrenosem, or other so-called hemostatic drugs, or the omission of aspirin will reduce the incidence of postoperative bleeding below that figure to be expected with careful surgical procedures alone.

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DISCUSSION

G. O. Proud, M.D., Kansas City, Kansas: The complication of hemorrhage following the resection of lymphoid tissue from the upper respiratory tract has long posed a grim challenge to the otorhinolaryngologist. In spite of the fine critical analysis which Drs. Proctor and Douglass have made of their own work, they would be the first to admit that the problem has not been resolved.

It was easy to find agreement with the speaker's technique, for the Crowe-Davis procedure has made possible better control of the patient and more meticulous surgical technique. Furthermore, it was gratifying to note that at least one-half hour was consumed in each operation; an operation such as this is deserving of sufficient time to provide for its completion. Statistics from the Massachusetts General Hospital, showing no correlation of bleeding or clotting time with incidence of hemorrhage, gave justification for the authors to ignore these laboratory steps. The clinical observation that blood transfusions seem to aid in bleeding control has again been recorded, and one is driven to wonder why this measure is not more frequently employed.

If one were to find fault with the material presented, it would lie in the indications for operation, for cough is not a symptom to be relieved by either tonsillectomy or adenoidectomy. Cervical adenitis, recurring upper respiratory infections, and pyrexia of unknown origin are also conditions which scarcely warrant such surgical intervention. Furthermore, the ratio of 7 per cent adenoidectomy to 93 per cent tonsillectomy seems to be somewhat out of proportion. Ten per cent of the procedures were carried out to relieve conductive deafness in children. Wehrs has shown that such deafness is almost invariably caused by middle ear effusion, and he advocated myringotomy for removal of the fluid in every instance. He suggested that adenoidectomy alone relieved only those patients who had suffered from the condition for a short time, for in these the fluid was thin enough to pass through the eustachian tube.

That fourteen post-quinsy patients had no bleeding was not surprising, because thrombosis of the blood vessels surrounding a peritonsillar abscess is the rule. Dr. Proctor is to be congratulated on his low incidence of hemorrhage, for only one out of 100 bled significantly and one of 275 bled severely. It is probable that anyone doing this operation will feel that his incidence is infinitely higher, particularly during the moments while he is endeavoring to control the bleeding.

The suggestion that bleeding inhibitors are without value and careful technique is the best preventative deserves support.

The report by one observer of four deaths in 20,000 operations should impress one with the seriousness of these surgical procedures and cause one to give careful consideration to their indications.

In Memoriam

August Leo Beck.....New Rochelle, New York
July 5, 1958

Daniel W. Brickley.....Marion, Ohio
April 15, 1958

Clyde Robert Cowan.....Buzzards Bay, Massachusetts
May 10, 1958

Elmo Eugene Dillon.....Homewood, Illinois
May 13, 1958

Edward Philip Drescher.....Berkeley, California
July 12, 1958

Harry H. Haggart.....Cincinnati, Ohio
July 13, 1958

William Jacob Hertz.....Allentown, Pennsylvania
May 4, 1958

Chevalier Jackson.....Schwenkville, Pennsylvania
August 16, 1958

H. F. Minor.....Memphis, Tennessee

Daniel Cameron Montgomery.....Greenville, Mississippi
May 29, 1958

Max Nisselson.....New York, New York
May 12, 1958

Robert Linton Ralston.....Spartanburg, South Carolina
April 26, 1958

Olga L. Sitchevska.....New York, New York
May 20, 1958

Charles Alston Thigpen.....Montgomery, Alabama
April 23, 1958

Gabriel Tucker.....Rosemont, Pennsylvania
April 17, 1958

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INSTRUCTION

"The object of this Academy shall be to promote and advance the science and art of medicine appertaining to the eye, ear, nose and throat; and to encourage the study of the relationship of these specialties to surgery, general medicine and hygiene." *(Constitution and By-Laws, American Academy of Ophthalmology and Otolaryngology, Article II)*

Prof. Victor C. Vaughn, while dean of the School of Medicine, University of Michigan, annually gave a course of lectures to freshmen medical students on the subject of hygiene. At the first lecture he would say, "The subject of this course of lectures is Hygiene. Hygiene is anything I choose to talk about." His lectures covered a wide range of subjects and gave the students their first intimation of the extent of the field of medicine, the responsibility of the physician in matters of public health, personal hygiene, science and education. Doctor Vaughn was a noted teacher and administrator, the leader of a faculty of experienced scientists who spent their entire lifetime in academic pursuits in the laboratories and classrooms. Not the least of their accomplishments was achieved in the classroom. Graduate students in basic sciences during Doctor Vaughn's period were comparatively few. Undergraduate students often doubled as laboratory assistants for a year or two, then went back to curricular assignments to obtain the M.D. It was regarded as highly advantageous for every professor in the medical school to have an M.D. degree. Other degrees —A.B., M.S., M.A., and Ph.D.—were earned in the fields of science, usually before entering medical school. Postgraduate work in clinical medicine did not lead to a college degree. The "campus faculty" were full-time teachers first, then researchers to whatever extent time and facilities were to be found. Didactic lectures were supplemented by textbooks, occasionally by lantern slides, and manuals for canned laboratory experiments in chemistry and physiology. It was a stiff program for a student with a weak memory but easy sailing for one with a retentive memory and a flair for recitation or writing.

Today the national medical societies are made up mostly of practitioners

who are primarily interested in advancing themselves in the art and science of their chosen profession. The benefit to the public through better medical service is inherent but not dominant. There is no fanatic zeal among physicians. Hence, there is no dogmatism, no cultism, no fads, no schisms to weaken the structure built on scientific enterprise as we recognize it and adhere to it today.

The Academy is in its sixty-third year. Scientific assemblies have been held in each year, except in 1945 when meetings were discouraged during the war. In 1921 the First Postgraduate Instructional Course was conducted by the Postgraduate Committee. There were 37 lecturers, all from medical school faculties and including deans, professors and instructors. The Postgraduate Committee has since been supplanted by Secretaries for Instruction, but the instruction courses have grown to be the chief attraction at the annual meeting. Emphasis on teaching placed the standard of scientific programs at the peak of medical intelligence, and the scientific programs of the Academy have always been instructive. Many of the teachers' manuals published by the Academy for Home Study Courses have grown from original presentations on the Scientific Program or in the Instructional Courses.

The Home Study Courses, begun in 1940 for residents and for refresher and reviews by the older practitioners, are now used in the medical schools of all states and territories, in the United States Armed Forces, and in several foreign countries in Europe and South America. The Academy has produced motion pictures and has published manuals, atlases of pathology, and periodicals that were not available from commercial agencies, all for the purpose of promoting the educational and instructional objectives of the Academy.

The faculties of the Academy's instruction and education departments have employed the methods of teaching practiced in the schools where they teach. At the annual meeting several hundred lecturers observe the work of their colleagues. This has resulted in a marked improvement in the lectures given at the convention and has raised the level of instruction at home as well. It is evident in the results of examinations by the certifying specialty boards. The Academy faculties are now conscious of the need of more trained teachers and of the desirability of improvement in teaching methods.

The Teachers' Sections have planned a joint meeting at 9:00 a.m. on Sunday, October 12, in the Grand Ballroom in the Palmer House. Three lecturers in the field of professional pedagogy have been invited to speak. Whatever they choose to talk about is Hygiene. Members and teachers are cordially invited to attend. Due to the early hour of the meeting the badge rule indicating registration will be waived.

THE TECHNIQUE FOR ACQUIRING AND PREPARING THE HUMAN TEMPORAL BONE FOR PATHOLOGIC STUDY AND FOR ANATOMICAL-SURGICAL DISSECTION

THE acquisition of fresh human temporal bones has become an essential adjunct to the training and research programs of departments of otolaryngology.

It is of utmost importance that temporal bones in a good state of preservation be acquired for pathologic study from patients who had suffered from hearing and/or vestibular disorders. Specimens are needed, for example, to show the pathologic changes in postural vertigo, congenital deafness, sudden deafness, streptomycin ototoxicity, and many other disorders.

Another important reason for acquiring temporal bones is the need for surgical experience. Otologic surgical procedures now are performed almost entirely with the aid of the operating microscope, and considerable practice is required to manipulate delicate instruments in this magnified visual field. Such skill can be acquired only by anatomic dissection and surgical exercises on the fresh human temporal bone.

There is an abundant supply of fresh human temporal bones in the autopsy room. Ordinarily, when permission for brain study has been granted, the temporal bones can be removed routinely. The brain should be removed with care so that the eighth cranial nerve is cut flush with the orifice of the internal auditory meatus; thus, a portion of the nerve trunk remains within the temporal bone specimen.

Prepared and submitted by the Subcommittee on Research of the Committee on the Conservation of Hearing, American Academy of Ophthalmology and Otolaryngology.

The temporal bone may be removed with the mallet and chisel, but care must be exercised to avoid fracturing it and avulsing the skin of the external auditory canal. Motor driven circular saws of a suitable size may be used but have the disadvantage of being dangerous to handle. The oscillating saw is the most effective instrument for this purpose and may be used with either the rocker type of saw blades or bone plug cutters (fig. 1).

FIG. 1—The outline for block removal is shown on the left and for bone-plug removal on the right.

The block method, which can be performed with the chisel and mallet, circular saw or oscillating saw with flat blade, will be described first.

I. TECHNIQUES FOR REMOVING TEMPORAL BONES

1. *Block Method*

Cuts 1 and 2 (fig. 1) are made approximately at right angles to the pet-

rous pyramid. Cut 1 is made near the apex of the petrous bone. The farther forward this cut is made, the larger is the portion of the eustachian tube obtained for study. Cut 2 is made about parallel to cut 1 and near the lateral wall of the middle cranial fossa. It is not necessary to obtain all of the mastoid process. Cut 3 is made vertical to the floor of the middle cranial fossa, about one inch in front of the petrous ridge. Cut 4 is an undercut of the block of tissue obtained by the preceding three cuts. It is made in the anatomically horizontal plane as near to the floor of the posterior cranial fossa as possible. Usually it is possible to make the cut pass through or below the dural exit of the vagus nerve.

Removal of the block outlined by the four saw cuts is often the most difficult part of the procedure. The block of tissue often is retained by some bony connections and by dense fibrous tissue of the mandibular articulation. Attempts to remove the block forcibly by the use of ordinary large forceps or by prying can result in crushing of pneumatized areas of bone or in fracturing through the middle ear cavity. The best instrument for removing the block is a "lion-jawed" forceps. The block is grasped by placing the two prongs of one jaw of the forceps in cut 4 and in closing down the other jaw in such a manner that its prongs grasp the superior surface of the block. With the block grasped as described above, gentle back and forth movement causes the unsevered bony connections to break without damage to important structures. After the bony connections are broken but while continuing to grasp the block with the forceps, it can be twisted enough to permit insertion of a knife along cuts 3 and 4 and severance of the dense fibrous connections remaining. A wide sharp chisel will often prove helpful, especially in severing the styloid process.[1]

2. *Bone Plug Method*

This preferred method consists of using bone plug cutters in the oscillating Stryker saw.* For the adult body, the proper saw diameter is 1.5 inches adjusted to a depth of 1.5 inches, whereas for the child and infant, the 1.0 inch and the 0.75 inch sizes are suitable. The cutter is centered on the arcuate eminence. Water is used as a lubricant to clear the fine cutting teeth of bone dust. Better cutting action is acquired by pivoting the saw back and forth through an arc of 90 degrees. A loss of resistance is sensed when the saw has penetrated the base of the skull. The plug is grasped with a bone forceps and the inferior attachments are cut with the knife, scissors or osteotome.†

With either method, the carotid arteries should be grasped and ligated and the stumps of the external auditory canals should be sutured tightly (figs. 2, 3 and 4).

When properly performed, removal of the temporal bone causes no mutilation or deformity of the ear or jaw.

The temporal bones should be removed by the otologist or a trainee in otolaryngology. The procedure should not interfere with the work schedule of the pathologist, increase the technical problems of the mortician, or change the external appearance of the body. Certain legal aspects, such as the necessity for acquiring special permission to remove the temporal bones, vary in different localities and with different interpretations of the law.

*The Stryker bone saw is available from the Orthopedic Frame Company, Kalamazoo, Michigan.

†Two motion pictures showing the techniques for removal of the temporal bones are available. One is a ten-minute 16 mm. silent film by Dr. Edmund Fowler, Jr., showing the block method of removal, and the other is a twenty-minute 16 mm. film with sound by Dr. Harold F. Schuknecht, showing the bone-plug cutter technique. These films may be acquired from the Executive Secretary of the American Academy of Ophthalmology and Otolaryngology.

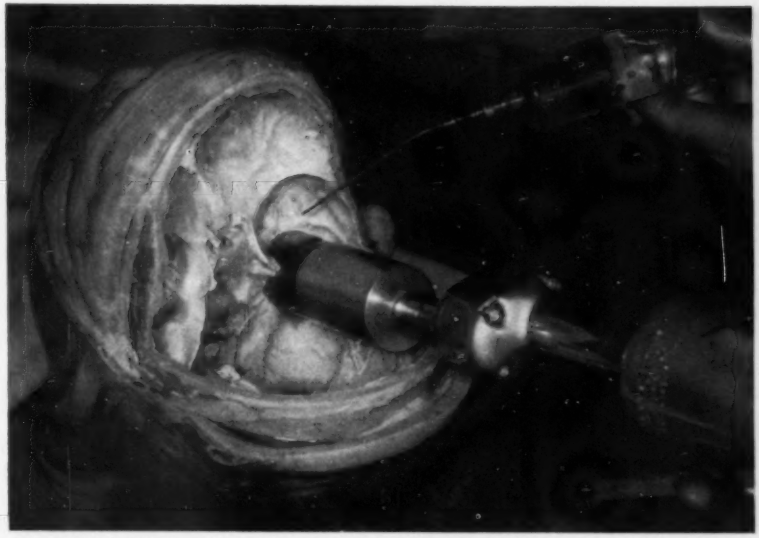


FIG. 2—The bone-plug cutter in position to remove the specimen from the right side.

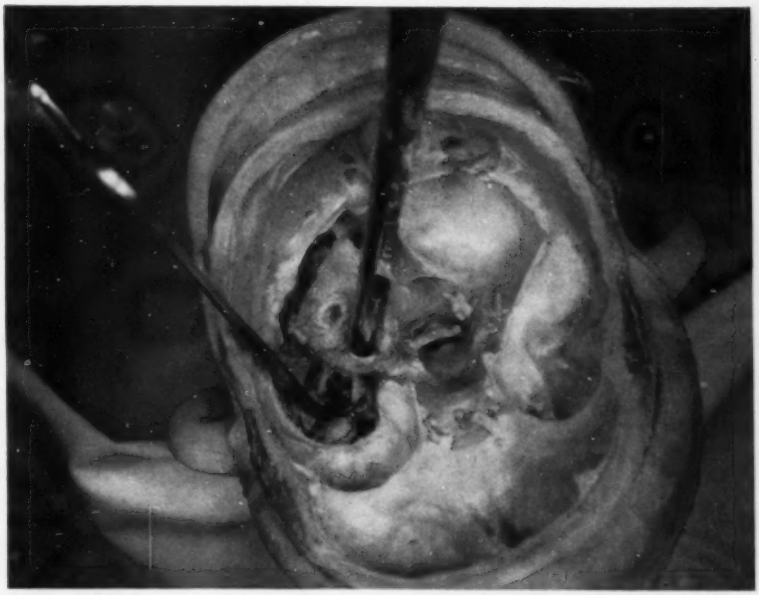


FIG. 3—The specimen has been rocked loose and the carotid artery grasped, preliminary to cutting and ligating.

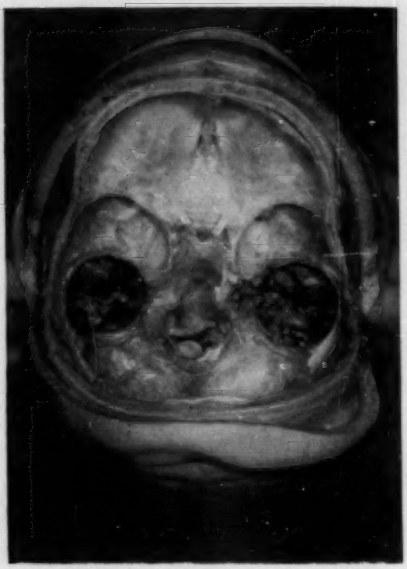


FIG. 4—The base of the skull after removal of both bones by the bone-plug cutter.

II. PREPARATION FOR HISTOLOGIC STUDY

When the temporal bones are acquired from patients who suffered from loss of hearing or vertigo, they should be prepared for histologic study. When it is possible to do so, audiometric and other hearing tests as well as vestibular tests should be acquired before death so that the pathologic changes can be correlated with function.

1. *Fixation Method*

Immediately after the bones have been removed, they should be placed into 400 cc. of 20 per cent formalin (one part commercial formalin to four parts water). In diluting the formalin, it is preferable to use water which has been degassed in vacuum because it will absorb air bubbles trapped in the middle ear and mastoid and will facilitate penetration of a fixing solution. The specimens are placed in fresh 10 per cent formalin solution each day for two days. The specimens can be kept indefinitely in formalin.

When the histopathologic changes in the inner ear are likely to be of primary importance, more rapid fixation can be obtained if the stapes is removed or subluxated. This is undesirable in cases in which the pathologic tissue changes about the stapes footplate are of primary importance. When permission for autopsy has been obtained early, fixation may be aided by the injection of the formalin solution into the middle ears immediately after death.

2. *Processing*

If an otologist or other person has acquired temporal bones suitable for sectioning and has no facilities for preparing them, arrangements for processing can be made directly with any one of a number of otological laboratories.‡

3. *Shipping*

After the bones have remained in 10 per cent formalin solution for three days or more, they may be shipped. They should be packed in an airtight jar with the intervening space occupied by absorbent paper or cotton. Excess fixative solution is poured off so that the absorbent paper or cotton holds only enough fixative to keep the bones moist.

4. *Distribution*

The Subcommittee on Research suggests that the cooperating laboratories prepare three sets of slides from each specimen submitted to them for histologic preparation. One set should be furnished to the otologist who provides the specimen, a second set may be retained in the laboratory, and the third set reserved for some central collecting laboratory (to be determined) where

‡A list of the cooperating otologic laboratories may be acquired from the Executive Secretary of the American Academy of Ophthalmology and Otolaryngology.

such specimens will be available for general study. The remaining unmounted sections should be retained by the cooperating laboratory to be available as needed for special stains.

5. *Information to Accompany Specimens*

It is suggested that certain information furnished with the bones be kept on file in the processing laboratory and a copy filed with the set of slides for the central collecting laboratory.

This should include identification, history of the case, otologic functional tests, diagnosis of terminal illness, time of death and time of placing bones in fixative, and the autopsy report.

When appropriate, the submitting otologist and the laboratory director should make an original report on the histopathologic findings. The accumulated specimens at the central collecting laboratory may be used for correlative studies by interested persons.

III. PREPARATION FOR ANATOMICAL-SURGICAL DISSECTION

Those specimens which are to be used for future anatomical-surgical dissection may be preserved by refrigeration. They can be placed in a jar containing cotton soaked in 1:1000 aqueous metaphen and stored in the cooling compartment of the refrigerator. The fresh state may be preserved for many months by placing the specimens in moist cotton, wrapping them in freezer paper and placing them in the freezing compartment.

A temporal bone specimen may be prepared for anatomical-surgical dissection by embedding it in plaster of Paris in a suitable container. A Frenckner

FIG. 5—The Frenckner temporal bone holder.

temporal bone holder,§ which consists of a heavy hemispherical bowl held in a stand which permits it to be tilted, is recommended (fig. 5). In order to make the most use of the anatomic material, the trainee must first acquire a detailed knowledge of the anatomy of the temporal bone. He should also have available a full set of surgical instruments, including head light, loupe, irrigation equipment, suction apparatus, bone drill and operating microscope.

§The Frenckner temporal bone holder is available from the Douglas Tool Company, 2300 East Nine Mile Road, Hazel Park, Michigan.

REFERENCE

Guild, Stacy R.: Technic for removing and preparing temporal bones for microscopic study. In Medicine of the Ear, edited by Edmund P. Fowler, Jr., New York, Thomas Nelson, 1947, pp. 663-668.

SIXTY-THIRD ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

OCTOBER 12 TO 17, 1958

PALMER HOUSE

Chicago, Illinois

OFFICIAL NOTICES

Convention Call

Under the authority of the Constitution and with the approval of the Council, I herewith proclaim that the Sixty-Third Annual Session of the American Academy of Ophthalmology and Otolaryngology will convene at the Palmer House, Chicago, Illinois, on Monday, October 13, 1958, at 9:30 a.m., and will continue through Thursday, October 16, 1958.

LEROY A. SCHALL, M.D.
President

Attest:
WILLIAM L. BENEDICT, M.D.
Executive Secretary-Treasurer

Business Meeting

The Annual Business Meeting of the Academy will be held in Private Dining Room 18 of the Palmer House on Thursday, October 16, 1958, at 5:30 p.m.

The Order of Business:

1. Call to order
2. Report of Executive Secretary-Treasurer
3. Report of the Senior Member of the Council
4. Election of Fellows
5. Election of Officers
6. Unfinished business
7. New business

All Fellows and candidates for election to Fellowship are urged to attend.

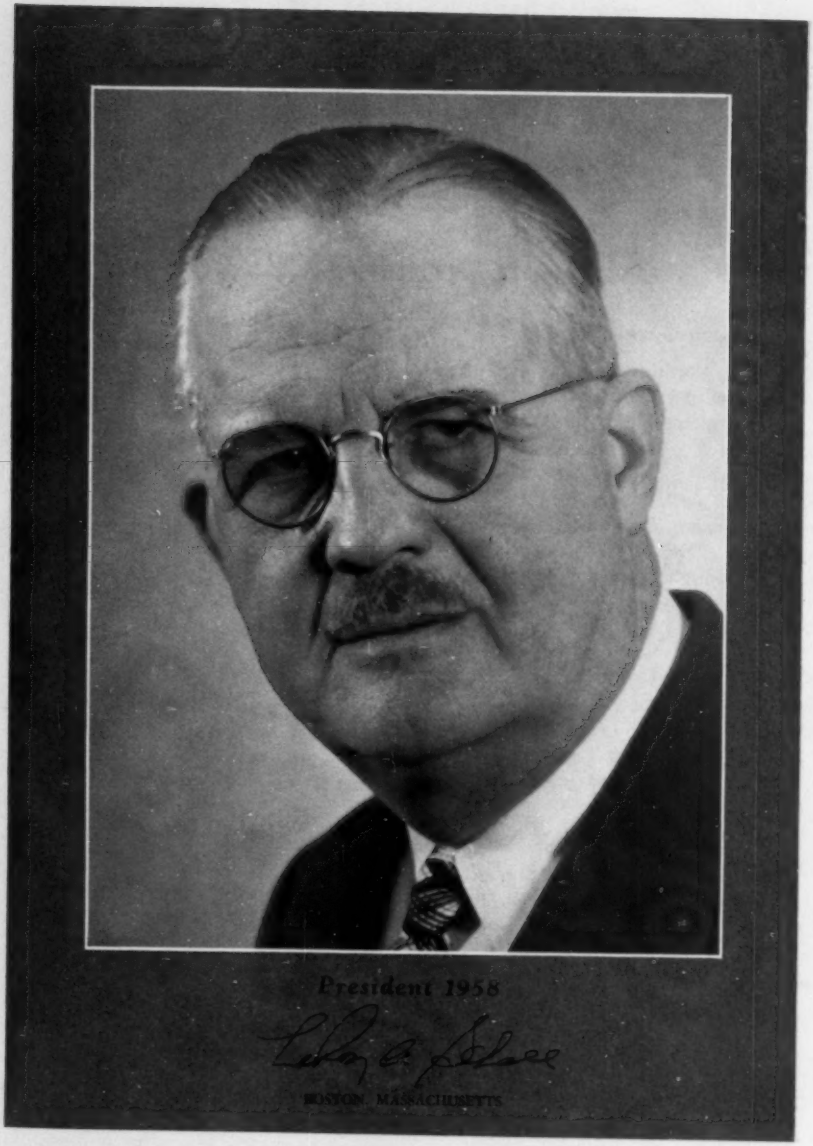
Council Meetings

The Council of the American Academy of Ophthalmology and Otolaryngology will convene at nine o'clock, Saturday morning, October 11, 1958, in Private Dining Room 4 of the Palmer House, Chicago. There will also be a meeting of the Council on Wednesday, October 15, at 12:45 p.m. in Private Dining Room 5. Members desiring to be heard or to present business will confer with the Executive Secretary-Treasurer for appointments.

LEROY A. SCHALL, M.D.
President

New Business

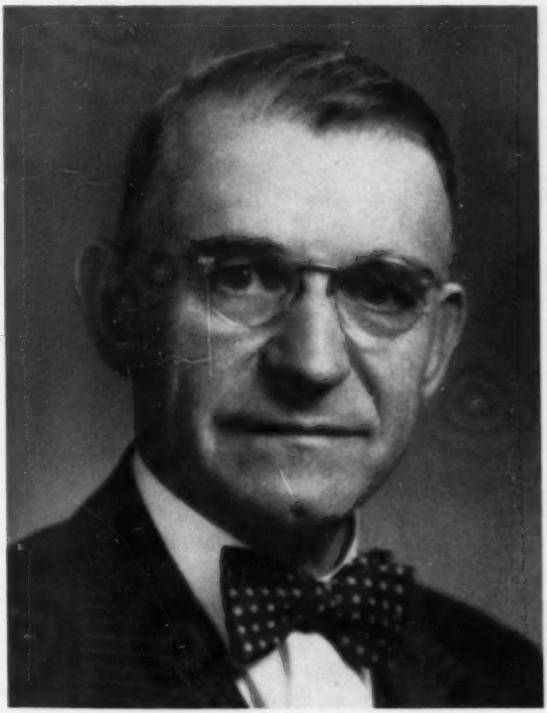
Proposals for new activities often are presented too late during the Academy session to afford time for deliberation. Therefore, new items requiring Council action should be presented in writing to



President 1958

BOSTON, MASSACHUSETTS

Guest of Honor



LOUIS HENRY CLERF, M.D.

PROFESSOR EMERITUS OF LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY, JEFFERSON MEDICAL COLLEGE OF PHILADELPHIA

Graduate of Jefferson Medical College of Philadelphia, 1912. Intern (1912-1914) and Chief Resident Physician (1914-1916), Jefferson Medical College of Philadelphia. Laryngologist, U. S. Navy, 1917-1920; Captain Medical Corps, U. S. Naval Reserve. Postgraduate Course in Bronchoscopy and Esophagoscopy, University of Pennsylvania School of Medicine, Philadelphia, 1922. Associate in Laryngology and Broncho-esophagology (1922); Professor of Laryngology and Broncho-esophagology (1930); Emeritus Professor of Laryngology and Broncho-esophagology (1954), Jefferson Medical College of Philadelphia.

Life Fellow of the American Academy of Ophthalmology and Otolaryngology (1923) and First Vice-President in 1957. Secretary (1927), President (1931), and Editor (1939) of the American Broncho-Esophagological Association. Secretary (1947) and President (1952) of the American Laryngological Association. President (1950) of the American Laryngological, Rhinological and Otological Society. Past Chairman and Secretary of the A.M.A. Section of Laryngology, Otology and Rhinology. Fellow of the American College of Chest Physicians; of the American College of Physicians; and of the American College of Surgeons. Member of the American Association of Thoracic Surgery; and of the American Therapeutic Society.

the Executive Secretary-Treasurer early so that the Committee concerned may have an opportunity to study the matter prior to presentation to the Council.

Public Relations

Contributors to the scientific program are requested to send copies of their papers to the Secretary for Public Relations, Glen G. Gibson, M.D., 2031 Locust Street, Philadelphia 3, at the earliest possible moment.

Exhibits

The Scientific and Technical Exhibits will open at nine o'clock on Sunday morning, October 12. They will close at four o'clock Sunday afternoon for the President's Reception. From Monday through Friday they will open at 8:30 a.m. The Scientific Exhibit will close at 5:30 p.m. each day, and the Technical Exhibit at 5:00 p.m., except that they will both close on Friday, October 17, at twelve o'clock noon.

Dues

Receipt card for 1958 dues is required for registration at the Academy meeting. Those who have not paid dues for 1958 are requested to remit to W. L. Benedict, M.D., Executive Secretary-Treasurer, 15 Second Street S.W., Rochester, Minn. In case of loss, a duplicate receipt card may be obtained upon request.

Registration

General registration will begin at 8:15 a.m., Sunday, October 12, in the Foyer to the Grand Ballroom on the fourth floor of the Palmer House.

The regulation badge secured at the time of registration is essential for admittance to all activities. To facilitate registration the following sections will be provided:

Advance Orders: Members who have reserved instruction course tickets register here.

Members Without Advance Orders: Please present Receipt Card for 1958 dues. Dues outstanding may be paid at this section.

Instructors: All persons presenting instruction courses register here to secure special badge.

Nonmembers: A registration fee of $5.00 is required of all guests (see next page). The following exceptions are made:

1. Candidates for Academy Fellowship whose names appear in the July-August issue of the TRANSACTIONS.
2. Residents in training presenting guest cards obtained before the meeting (see next page).
3. Guest instructors.
4. Guests taking part in the Scientific Program or Exhibits.

Orthoptic Technicians: All orthoptic technicians register here. Tickets for the Orthoptic Technicians Instruction Courses will be on sale here.

Guests

Physicians who are not members are welcome to attend the meeting of the Academy as guests and to participate in all the activities. All guests, however, will be required to register, and a fee of $5.00 will be charged.

In the past, a number of Fellows and registered guests of the Academy have indicated that they wish their *office nurses, technicians,* and *similar personnel* to attend parts of the Academy program or exhibits. Such individuals will be allowed to register upon presentation of a written request from a Fellow or if accompanied by a registered guest or Fellow. The guest fee of $5.00 will be required. An exception is made for all members of the American Association of Orthoptic Technicians, who may register without fee.

Residents

Men in training in accepted residencies, or those now completing a full academic year (nine months) in basic study, may register without fee upon presentation of a guest card obtained by written request of their Department Heads.

Requests from Department Heads for guest cards should be sent to W. L. Benedict, M.D., 15 Second Street S.W., Rochester, Minn., by October 1.

Instruction Course Tickets

The tickets for courses in ophthalmology and the tickets for courses in otolaryngology and maxillofacial surgery will be sold at separate desks in the Registration area. Bulletin boards behind each desk will indicate what tickets are available. Please consult these boards in making your selections. Bear in mind that individual courses are for one hour only; continuous courses run for two or more hours. Price of tickets is $2.00 per hour.

It is necessary to register and to be wearing a regulation badge in order to purchase instruction course tickets.

Ladies' Registration

The wives of members and guests are urged to register at the Ladies' Headquarters on the eighth floor of the Palmer House, Room 832. Registration will open at nine o'clock, Sunday morning, October 12, and will close Wednesday evening, October 15.

Dinner Tickets

Tickets for the Annual Banquet, which will be held on Monday evening, will be on sale beginning Sunday morning at nine o'clock, in the Registration Area, and it is urged that purchases be made early. Ticket sale will close at two o'clock on Monday afternoon.

Tickets for Alumni Dinners on Wednesday evening will be on sale from Sunday morning at nine o'clock until two o'clock on Wednesday.

CONVENTION ACTIVITIES

PRESIDENT'S RECEPTION

Grand Ballroom

Sunday, 4:30 p.m.

President LeRoy A. Schall and Mrs. Schall invite all members and guests of the Academy to a reception on Sunday, October 12, at 4:30 p.m. in the Grand Ballroom.

XV JACKSON MEMORIAL LECTURE

Sponsored by the Ophthalmic Publishing Co.

Dr. Arthur Linksz, Assistant Professor of Ophthalmology in New York University College of Medicine, New York, will deliver the XV Jackson Memorial Lecture at the sicentific session of the Section on Ophthalmology, Tuesday morning, October 14. The subject of his lecture will be "Aniseikonia: With Notes on the Jackson-Lancaster Controversy."

XIV WHERRY MEMORIAL LECTURE

Sponsored by the Wherry Memorial Fund

The XIV Wherry Memorial Lecture will be delivered at the scientific session of the Section on Otolaryngology, Wednesday afternoon, October 15, by Dr. Julius Lempert, Research Professor of Otolaryngology at Tufts College Medical School, Boston; Surgical Director of the Lempert Institute of Otology and Research Director of the Lempert Research Foundation, New York. The subject of his address will be "Basic Principles Used in the Development of Modern Temporal Bone Surgery Technique."

THE ANNUAL BANQUET

Grand Ballroom

Monday, 7:00 p.m.

The attendance at the annual banquet last year was greater than for several years past and it was attributed to the good food and good entertainment at less than night club prices.

Tables may be reserved, ten places to a table, at the ticket box in the registration foyer until 2:00 p.m., Monday. Doors open at 6:30 p.m. Bar service available, dress optional.

FACULTY LUNCHEON

Private Dining Room 14

Thursday, 12:30 p.m.

Members of the Instruction Courses Faculty, the Scientific Exhibitors, and authors and discussers on the Scientific Programs are invited to a Buffet Luncheon in Private Dining Room 14, Thursday at 12:30 p.m.

LADIES' PROGRAM

Wives and guests of members of the Academy are invited to participate in the special program of events planned for their pleasure and convenience by the Academy.

Coffee and rolls will be served daily from 10:30 to 11:30 a.m. and from 4:00 to 5:00 p.m. in the Ladies' Hospitality Suite, Rooms 832-834. On Sunday afternoon the coffee hour will be from three to four o'clock.

Information personnel will be in attendance in the Hospitality Suite to give suggestions and advice regarding restaurants, shopping, theaters, concerts, and places of interest in the city.

SPECIAL LECTURES

Dr. Theodore Schmidt of the University Eye Clinic in Berne, Switzerland, will give a lecture on APLANATION TONOMETRY on Monday, October 13, 1958, at 3:15 p.m. in Room 710 at the Palmer House.

This lecture is free of charge and tickets may be obtained at the instruction desk for ophthalmology in the registration area. If there is sufficient demand, the lecture will be repeated on Thursday, October 16, at 2:00 p.m. in the Crystal Room on the third floor of the Palmer House.

ALUMNI DINNERS — 1958

The following alumni groups will hold dinners, luncheons, or cocktail parties during the Academy meeting. Most of these functions will be held on Wednesday evening, October 15, 1958. (A complete listing including time, place of function and address of the secretary for each group will appear in the Pocket Program issued at the Academy meeting.)

Brooklyn Eye and Ear Alumni Association

Dr. J. Mackenize Brown Alumni

Buffalo Alumni

Cook County Hospital Ophthalmological and Otolaryngological Alumni

Episcopal Eye, Ear & Throat Hospital Residents Alumni

Henry Ford Hospital Alumni

Gill Memorial Eye, Ear and Throat Hospital Alumni—Annual Spring Graduate Course

Grady Hospital—Emory University Alumni

Harper Hospital Alumni

Heed Ophthalmic Foundation Alumni

Dr. Gordon D. Hoople Alumni

Illinois Eye and Ear Infirmary Alumni — Centennial Dinner

Mail advance reservations to
Mr. Lester R. Gerber, Superintendent,
Illinois Eye and Ear Infirmary,
904 W. Adams St.,
Chicago 7, Ill.

Indiana Alumni and Staff

Iowa Eye, Ear, Nose and Throat Alumni

Johns Hopkins Hospital Alumni—Wilmer Institute

David H. Jones Alumni

Dr. Arlington C. Krause Luncheon

Dr. John R. Lindsay Alumni

Manhattan Eye, Ear and Throat Hospital—Society of Ex-House Surgeons

William A. Mann Ophthalmological Alumni of V. A. (Hines, Ill.)

Massachusetts Eye and Ear Infirmary and Harvard Medical School Alumni

Massachusetts Eye and Ear Infirmary, Retina Service Alumni

Mayo Clinic Alumni

Michael Reese Hospital Eye Alumni

Mount Sinai Hospital of New York—Otolaryngology Alumni

Newark Eye and Ear Infirmary Alumni

New York Eye and Ear Infirmary Alumni

New York Hospital Alumni Group

New York University, New York Post Graduate Medical School and Bellevue Hospital Alumni

Northwestern University Alumni, Postgraduates, Residents and Staff

Walter R. Parker-Roy Bishop Canfield Alumni

Presbyterian Hospital of New York Ear, Nose and Throat Alumni

Receiving Hospital Ophthalmic Alumni (Detroit)

University of Pennsylvania Post-Graduate (Ophthalmology) and Wills Hospital Alumni

University of Pennsylvania Alumni—Otolaryngology

Washington University Alumni—Ophthalmology

Washington University Alumni—Otolaryngology

Wisconsin Eye, Ear, Nose and Throat Alumni

CANDIDATES--1958

The following candidates have qualified for Fellowship in the American Academy of Ophthalmology and Otolaryngology and will be voted upon at the Annual Business Meeting, Thursday, October 16, 1958. Objections to the election of a candidate must be filed with the Executive Secretary-Treasurer previous to Saturday, October 11, 1958.

CANDIDATES CERTIFICATED BY BOARDS

Ophthalmology (OP)
Otolaryngology (ALR)
Plastic Surgery (Pl S)

Altfather, Ellis McFerrin, Ireland Army Hospital, Fort Knox, Ky., OP
Anderson, Robert Garfield, 2760 W. Foster Ave., Chicago 25, Ill., OP
Anthony, James McCullough, 8215 Westchester Dr., Dallas 25, Texas, ALR
Anthony, Marc, 508 Old Nat'l Bank Bldg., Spokane 1, Wash., OP
Arribas, Neva P., 1612 Rizal Ave., Manila, Philippines, OP
Arsenault, Armand, 1150 E. St. Joseph Blvd., Montreal, Que., Canada, ALR
Ashcraft, Elijah Jefferson, Jr., 613 W. Filmore St., Harlingen, Texas, ALR
Azar, Robert F., 1123 Maison Blanche Bldg., New Orleans 16, La., OP

Baade, Ernest Alfred, 33 W. Third St., Lewistown, Pa., ALR
Barnes, Charles Rupert, 3720 Washington Blvd., St. Louis 8, Mo., OP
Bartholomew, Henry Homer, 508 E. South Temple St., Salt Lake City 2, Utah, OP
Beck, Paul M., Kirkpatrick Bldg., St. Joseph, Mo., OP
Behle, Charles F., 1046 E. First South St., Salt Lake City 2, Utah, OP
Belzile, Marcel J., 936A Sherbrooke St. East, Montreal, Que., Canada, ALR
Bennett, Truett Vann, 144 Ponce de Leon Ave. N. E., Atlanta 8, Ga., ALR
Bercovitz, Jean T., 3465 Cote des Neiges, Montreal 25, Que., Canada, ALR
Berk, Myles M., 4101 Jenkins Arcade, Pittsburgh 22, Pa., OP
Best, Ray Golding, 405 Pelissier St., Windsor, Ont., Canada, OP
Black, Henry H., 4062 Jenkins Arcade, Pittsburgh 22, Pa., OP
Blais, Raymond, 837 Cherrier St., Montreal, Que., Canada, ALR
Blatt, Irving Myron, Hauser Clinic, 7411 Third Ave., Detroit 2, Mich., ALR
Bock, Rudolf H., 145 N. California Ave., Palo Alto, Calif., OP
Bourne, Reginald Alan, 1414 Drummond St., Montreal 25, Que., Canada, OP
Brault, Jules, 418 Sherbrooke St. East, Montreal, Que., Canada, OP
Brockhurst, Robert John, 99 W. Cedar St., Boston 14, Mass., OP
Brown, James Stinson, 508-509 Greyhound Bldg., Calgary, Alta., Canada, ALR
Brown, John Joseph, 63 N. Common St., Lynn, Mass., ALR
Bunker, Rex James, 605 Professional Bldg., Santurce, Puerto Rico, ALR
Burton, Truman Young, 616 S. Tejon St., Colorado Springs, Colo., OP
Butler, William Ernest, 1222 N. 27th St., Billings, Mont., OP
Byers, Jerome Leon, 3607 Gaston Ave., Dallas 10, Texas, OP

Cacchillo, Rose C., 57 W. 57th St., New York 19, N. Y., OP
Carroll, John Dennis, Jr., 406 Fulton St., Troy, N. Y., OP
Carter, Robert Clare, 1322 Malgren Ave., San Pedro, Calif., OP
Caygill, Wayne Metcalf, 357 30th St., Oakland 9, Calif., OP
Chatterley, J. Garth, Medical Arts Bldg., Salt Lake City 11, Utah, OP
Christoferson, Kent William, 505 Eugene Medical Center Bldg., Eugene, Ore., OP
Cleasby, Gilbert William, 2400 Clay St., San Francisco 15, Calif., OP
Cook, Robert Dean, 220 Meridian Rd., San Jose, Calif., OP
Cooke, Samuel Leonard, U. S. Army Hospital, Fort Bragg, N. C., ALR
Culbertson, Marvin Criddle, Jr., 3619 Fairmount St., Dallas 19, Texas, ALR
Curts, Calvin Jeffries, 1124 Professional Bldg., Kansas City 6, Mo., OP

Dale, David O., 132 S. Prospect Ave., Park Ridge, Ill., ALR
Davis, Herbert A., 2900 S. Fort St., Detroit 17, Mich., ALR
Davis, Robert James, 305 Medical Center Bldg., Spokane 4, Wash., OP
De Francois, Walter, 122 S. Michigan Ave., Chicago 3, Ill., OP
de la Vega, Felix, 222 W. Exchange, Freeport, Ill., OP
Derivaux, Joseph H., The Street Clinic, Vicksburg, Miss., OP
Dimun, Michael Francis, 8078 Jenkins Arcade, Pittsburgh 22, Pa., OP
Dobson, Clare William, Ste. 213, 170 St. George St., Toronto 5, Ont., Canada, ALR
Dodd, Elbert W., Jr., 1726 M St. N. W., Washington 6, D. C., OP
Dole, John Addis, Jr., 513 Neal Ave., Ironton, Ohio, OP
Donn, Anthony, 635 W. 165th St., New York 32, N. Y., OP
Donohoo, Albert Curry, Brooke Army Hospital, Fort Sam Houston, Texas, ALR
Dunn, Ralph Orace, 714 Medical Towers, Houston 25, Texas, OP
Durk, Irving, 135 Ocean Ave., Brooklyn 25, N. Y., OP

Eckdahl, Robert Wilson, 1053 R St., Fresno, Calif., OP
Edwards, Larry Edwin, 1648 Post Rd., Fairfield, Conn., ALR
Egleston, DuBose, University of North Carolina Medical School, Chapel Hill, N. C., OP
Elliott, Dean C., Burns Clinic, Petoskey, Mich., ALR
Elsen, John, 636 Church St., Evanston, Ill., ALR
Elser, Otto H., 10901 Winner Rd., Independence, Mo., OP
Erickson, Raymond Leroy, 901 Livesley Bldg., Salem, Ore., OP

Fairbanks, Bryce Jay, 34 S. Fifth East, Salt Lake City 2, Utah, OP
Fairfax, Walter A., Jr., USAF Hospital, Sheppard AFB, Texas, OP
Fanjoy, Roy Wilfred, 8 Germain St., St. John, N. B., Canada, ALR
Felt, Robert S., 312 Medical Arts Bldg., Salt Lake City 11, Utah, OP
Fercho, Calvin K., 812 Black Bldg., Fargo, N. D., OP
Ferguson, Edward Clifton, III, University Hospitals, Iowa City, Iowa, OP
Fischer, Wolfram George, 645 W. Harding Way, Stockton 4, Calif., ALR
Fisher, George W., Jr., McPherson Hospital, Durham, N. C., OP
Flanagan, Clair Mills, 80 Sheboygan St., Fond du Lac, Wis., ALR
Flanary, Lemuel Madison, 530 W. 20th St., Houston 8, Texas, OP
Freeman, Richard Jerome, 14900 Detroit Ave., Lakewood 7, Ohio, ALR
Fuhring, Shirley Anthony, 117 E. Cook St., Santa Maria, Calif., OALR

Gahagan, Lawrence Oley, Lackland Air Force Base, San Antonio, Texas, OP
Gambacorta, Humbert Michael, 19 Holly St., Cranford, N. J., OP
Gitter, Joseph, 640 Main St., Malden 48, Mass., ALR
Glew, William Bainbridge, Washington Clinic, Wisconsin and Western Avenues, Washington 15, D. C., OP
Glueck, Sidney John, 34 W. High St., Springfield, Ohio, OP
Goodwin, Maurice Roy, 724 Pelham Parkway South, New York 62, N. Y., ALR
Greenberg, Richard Saul, 1421 Dodge St., Omaha 2, Neb., OP

Hagens, Walter Edward, 30 N. Michigan Ave., Chicago 2, Ill., OP
Halberg, G. Peter, 936 Fifth Ave., New York 21, N. Y., OP
Hara, Kinge, 1729 S. 11th St., Milwaukee 4, Wis., ALR
Harris, William S., 3616 Maple Ave., Dallas 19, Texas, OP
Helmick, Ernest D., 228 N. Second St., Harrisburg, Pa., OP
Hendrick, Robert Smith, Highland Clinic, Shreveport, La., ALR
Holland, Malvern Carlyle, U.S.P.H.S. Hospital, 4141 N. Clarendon St., Chicago, Ill., OP
Holm, Arvid Gustov, 2040 W. Wisconsin Ave., Milwaukee 3, Wis., OP
Hopen, Joseph Martin, 421 N. Federal Hghwy., Hollywood, Fla., OP
Hopkins, Robert Edgar, 242 N. Sutter St., Stockton 2, Calif., OP
Hoyt, William Fletcher, 384 Post, San Francisco, Calif., OP
Hungerford, Louis Newell, Jr., 1118 Ninth Ave., Seattle 1, Wash., OP

Iser, Gilbert, 3403 W. Lawrence Ave., Chicago 25, Ill., OP

Jackson, James Wishart, Winnipeg Clinic, Winnipeg 1, Man., Canada, ALR
Jackson, Thomas Marion, 1049 Madison Ave., Memphis 4, Tenn., ALR
Jennings, David Thorington, 912 W. Fourth St., Williamsport, Pa., OP
Johnson, Douglas L., Medical Center, Little Falls, Minn., OP
Johnson, Nicholas J., 2010 E. 102nd St., Cleveland 6, Ohio, OP
Johnson, William Rayner, 117 W. Boscawen St., Winchester, Va., OP
Jones, George Herbert, 3332 Convention St., Baton Rouge 6, La., OP
Jones, Samuel Thomas, 904 W. Adams St., Chicago 7, Ill., OP
Joseph, Donald John, Fitzsimons Army Hospital, Denver 8, Colo., ALR
Jungschaffer, Otto H., Massachusetts Eye and Ear Infirmary Retina Service, Boston 14, Mass., OP

Kaplan, Gerald, 8 Summit Ave., Hackensack, N. J., ALR
Karutz, Wallace S., 118 Pennsylvania Ave., Brooklyn 7, N. Y., ALR
Kavanagh, Thomas W., 612 Michigan Nat'l Bank, Battle Creek, Mich., ALR
Kelly, William James, 10 Union Ave., Lynbrook, N. Y., OP
Kemper, Robert Allen, 1308 Union Central Bldg., Cincinnati 2, Ohio, OP
Keskey, George Richard, 1714 E. Capital Dr., Milwaukee 11, Wis., OP
Kinkella, Albert Matthew, 650 S. Central Ave., Marshfield, Wis., ALR
Klotz, Robert E., 243 Charles St., Boston 14, Mass., ALR
Knight, Robert E., 705 N. East St., Bloomington, Ill., ALR
Kohl, Justin William, 2720 Capitol Ave., Sacramento, Calif., OP
Kraft, James Edward, 108 W. Sixth St., Tulsa 19, Okla., OP
Krimmer, Burton Mark, 5736 W. North Ave., Chicago, Ill., OP
Kuhn, Arthur J., 112 Rimbach St., Hammond, Ind., ALR
Kupersmith, Harry S., 117 N. Claudina, Anaheim, Calif., OP
Kurtsman, Joseph Louis, U. S. Naval Hospital, Beaufort, S. C., OP

Lakritz, Amrum, 30 N. Michigan Ave., Chicago 2, Ill., OP
Lanning, Charles D., 975 Ryland, Reno, Nev., OP
Lassiter, Lawrence H., 122 S. Michigan Ave., Chicago 3, Ill., OP
Lava, Irving M., 628 S. San Vicente Blvd., Los Angeles 48, Calif., OP
Lawrence, Carteret, Johns Hopkins Hospital, Baltimore 5, Md., OP
Lerman, Sidney, 260 Crittenden Blvd., Rochester 20, N. Y., OP
Levene, Ralph Zalman, 550 First Ave., New York 16, N. Y., OP
Lewis, John E., 24 Pine St. South, Timmins, Ont., Canada, OP
Light, Arthur, 25 E. Washington St., Chicago 2, Ill., OP
Lilly, Frederick Vivan, 407 Bair Bldg., Beckley, W. Va., ALR
Lineback, Carl Mayo, U. S. Army Hospital, Fort Chaffee, Ark., ALR
Locken, Gail Ann, 424 W. Woodruff Ave., Toledo 2, Ohio, OP
Lowry, Dick Moss, 1200 N. Walker, Oklahoma City 3, Okla., ALR
Lubrano, Daniel, 910 Citizen's Bldg., Tampa, Fla., ALR

MacDuffee, Frederic D., 34 N. Ash St., Ventura, Calif., OP
MacGlashan, Charles B., Jr., 600 S. Main St., Salinas, Calif., OP

Maier, Godfrey George, 1114 Heyburn Bldg., Louisville 2, Ky., ALR
Mamo, Jubran G., 1601 Spring Garden St., Philadelphia 30, Pa., OP
Martinez, Daniel McNamara, 1511 N. Beckley, Dallas 3, Texas, ALR
Masson, James Knowles, 200 First St. S. W., Rochester, Minn., Pl S
McArtor, James Richard, USN Hospital, Quantico, Va., OP
McCloskey, Robert Bruce, 745 E. Big Bend, Webster Groves 19, Mo., ALR
McCole, Cornelius Edward, Henry Ford Hospital, Detroit 2, Mich., OP
McDonald, Henry H., 127 N. Madison St., Pasadena, Calif., OP
McTigue, John William, 1746 K St. N. W., Washington 6, D. C., OP
Mettier, Stacy R., Jr., 490 Post St., San Francisco 2, Calif., OP
Miller, David Hugh, 22 Washington St., Cumberland, Md., OP
Miller, James E., 640 S. Kingshighway, St. Louis 10, Mo., OP
Miller, Robert Glasgow, 30 N. Michigan Ave., Chicago 2, Ill., OP
Miranda-Rivera, Manuel N., 720 Ponce de Leon Ave., Hato Rey, Puerto Rico, OP

Molzahn, Clifford Douglas, 1801 Carnegie Ave., Cleveland 15, Ohio, OP
Montgomery, Amzi Denbo, 905 Gen. Mouton Ave., Lafayette, La., ALR
Newby, William Edward, U.S.P.H.S. Hospital, Seattle 14, Wash., OP
Normann, Frank S., 1520 Aline St., New Orleans 15, La., ALR
Nursall, John F., 2163 Torrance Blvd., Torrance, Calif., OP

Ohta, Victor M., 2750th USAF Hospital, Wright-Patterson AFB, Ohio, OP
Okamura, Ichiro D., 99 West Cedar St., Boston 14, Mass., OP
Oleari, Valdo P., 214 N. Hale St., Wheaton, Ill., OP

Paine, Albert James, Bluefield Sanitarium Clinic, Bluefield, W. Va., ALR
Palmer, Robert Hamilton, Jr., U. S. Naval Hospital, Pensacola, Fla., OP
Patience, Hansi Rademacher, 940 Pine St., Glenview, Ill., OP
Penn, Robert F., V. A. Hospital, Hines, Ill., OP
Pennington, Claude Lee, 829 First St., Macon, Ga., ALR
Pernoud, Flavius G., 634 N. Grand Blvd., St. Louis 3, Mo., OP
Preston, Helen E., 15421 Sixth St., Victorville, Calif., OP
Pringle, Richard Warner, 9 E. Union St., Burlington, N. J., OP

Quilty, Thomas J., 112 E. Madison, Goshen, Ind., ALR

Raines, Donald Sterling, 32 N. Goodman St., Rochester 7, N. Y., ALR
Ramsey, Robert Bruce, 1414 Drummond St., Montreal, Que., Canada, OP
Reel, Charles M., Mellon Bank Bldg., New Kensington, Pa., OP
Rice, Marion Hobson, 542 Church St., Decatur, Ga., OP
Richards, Richard D., 721 Black Bldg., Waterloo, Iowa, OP
Richardson, Eric Carleton, 730 Fifth Ave., New York 19, N. Y., OP
Ritso, Olga, 11 Webster St., North Tonawanda, N. Y., OP
Rome, Albert, 101 W. North St., Danville, Ill., OP
Royce, Emery E., 123 S. Stone Ave., Tucson, Ariz., OP
Rumage, Joseph Paul, 709 Audubon Bldg., New Orleans 12, La., OP

Sade, Jacob, Department of Otolaryngology, Washington University, St. Louis 10, Mo., ALR
Schepens, Charles Louis, 99 W. Cedar St., Boston 14, Mass., OP
Schmidt, Ruth Berta, 1708 N. Garey Ave., Pomona, Calif., ALR
Schosheim, Arnold M., 1036 Lorimer St., Brooklyn 22, N. Y., ALR
Schultz, Marvin Harold, 114 N. Front St., Marquette, Mich., ALR
Scott, Alfred W., 7 Bay State Rd., Boston 15, Mass., OP
Scott, Pierre B., 1835 Eye St. N. W., Washington 6, D. C., OP
Seale, Hubert James, 1325 Hickory, Abilene, Texas, OP
Sergis, Mooshy, 127 E. Acacia St., Stockton 3, Calif., OP
Settel, Nathan, 10 W. 82nd St., New York 24, N. Y., ALR
Shamess, Sam Albert, 359 Queen St. East, Sault Ste. Marie, Ont., Canada, OP
Shankar, Ramaiyer Gouri, Ranjit Sadan, Station Rd., Jaipur, Rajasthan, India, ALR
Shapin, Sydney, 1346 50th St., Brooklyn 19, N. Y., ALR
Shea, John Joseph, 1018 Madison Ave., Memphis 4, Tenn., ALR
Shearer, Robert Vernon, 517 N. Central, Glendale 3, Calif., OP
Shepard, Marvin Glen, 722 Medical Arts Bldg., Dallas 1, Texas, ALR
Shramek, Joseph Martin, Veterans Administration Hospital, Long Beach 4, Calif., ALR
Shumate, Robert Edward L., 417 Cobb Bldg., Seattle 1, Wash., OP
Siedentop, Karl Heinrich, 715 Lake St., Oak Park, Ill., ALR
Silverstein, Murray Herman, 942 Hempstead Turnpike, Franklin Square, L. I., New York, N. Y., OP
Skloff, Samuel Harry, 1822 E. Allegheny Ave., Philadelphia 34, Pa., OP
Smith, Ruby A., 513 N. Charles St., Baltimore 1, Md., OP
Snider, Louis R., 133 E. 4800 South, Murray, Utah, OP
Sorokanich, Vera, First Nat'l Bank, Scranton, Pa., ALR
Spillane, James Henry, 379 S. Main St., Phillipsburg, N. J., ALR
Spradling, Richard Lee, 530 N. 27th St., Corvallis, Ore., OP
Statton, Roy F., 702 Sharpe Bldg., Lincoln 8, Neb., OP
Steinmetz, Charles Gordon, III, 4606 Spruce St., Philadelphia 39, Pa., OP

Stewart, Robert Bell, 221 Professional Bldg., Springfield, Mo., OP

Stock, Maurice Frederick, 319 Globe Bldg., Pittsburg, Kan., ALR

Stone, Sidney Lawrence, 14620 E. Seven Mile Rd., Detroit 5, Mich., OP

Sudarsky, Raymond David, 11 E. 68th St., New York 21, N. Y., OP

Summerlin, Jack Donald, Indiana University Medical Center, Indianapolis, Ind., ALR

Szewczyk, Edward John, 3939 State St., East St. Louis, Ill., OP

Talmage, Walter Raymond, 16 Elm St., Morristown, N. J., ALR

Teed, Roy W., V. A. Hospital, 921 N. E. 13th St., Oklahoma City, Okla., OP

Thomas, Francis T., 44 Pondfield Rd., Bronxville 8, N. Y., OP

Thornfeldt, Paul Richard, 919 Taylor St. Bldg., Portland, Ore., OP

Tippin, Ernest Elwood, Jr., 609 N. Broadway, Wichita 5, Kan., ALR

Travis, Willis, 90 S. Hamilton St., Poughkeepsie, N. Y., OP

Trimble, John Garfield, 116 S. Buckeye, Kokomo, Ind., OP

Trunzo, Thomas Harold, 207 Thompson St., Latrobe, Pa., ALR

Updegrove, Robert Albert, 428 Market St., Williamsport, Pa., ALR

Utrata, Joseph, 238 N. High St., Chillicothe, Ohio, ALR

VanArnam, Carleton E., 413 S. W. Fourth Ave., Gainesville, Fla., OP

VanDyke, Don Philip, 607 E. Main, Kent, Ohio, OP

Van Lonkhuyzen, Maurice, 31 Bramhall St., Portland, Maine, OP

Verity, Alden M., 248 W. Washington Blvd., Whittier, Calif., OP

Vernlund, Robert James, 85 Jefferson St., Hartford 14, Conn., OP

Vey, Edwin Kenneth, 355 Fifth Ave., Pittsburgh 22, Pa., OP

Vicens, Enrique A., Edificio Profesional 3A, Concordia 31, Ponce, Puerto Rico, ALR

Vidal-Gilet, Jose Humberto, Concordia 40, Ponce, Puerto Rico, OP

Wachs, Leonard V., 3650 Atlantic Ave., Long Beach 7, Calif., ALR

Wagenaar, Philip, Chanute Air Force Hospital, Chanute AFB, Ill., OP

Waldmann, Robert P., 5040 USAF Hospital, APO 942, Seattle, Wash., ALR

Wallace, Joseph, Jr., 5 N. Owen Ave., Lansdowne, Pa., ALR

Waller, Jesse Edward, 333 E. Huron St., Chicago 11, Ill., ALR

Walters, Florence Amelia, 2800 Milwaukee Ave., Chicago 18, Ill., OP

Wang, Hsin Fen, 280 W. MacArthur Blvd., Oakland 11, Calif., ALR

Warren, Claude M., Jr., Suite 101, 1720 Springhill Ave., Mobile, Ala., ALR

Webb, James William, 806 S. Church, Jonesboro, Ark., OP

Weisbaum, M. Byron, 215 S. Seventh St., Springfield, Ill., OP

Weisel, John Thomas, 836 E. Main St., Medford, Ore., OP

Werner, Nathan Harold, 309 Hargrove St., Winnipeg, Man., Canada, OP

White, Frederick C., 175 25th Street, Richmond, Calif., OP

Wilson, Robert B., 765 New Brotherhood Bldg., Kansas City 1, Kan., ALR

Wilson, William Errol, 1455 Waterman Ave., Suite 124, San Bernardino, Calif., OP

Winn, William E. Ted, Jr., 1300 Bancroft Ave., San Leandro, Calif., OP

Wittchow, Allen William, P. O. Box 734, Oshkosh, Wis., OP

Wold, Keith C., 409 Professional Bldg., Fort Lauderdale, Fla., OP

Wolferman, Adolph, 36 Greene St., Cumberland, Md., ALR

Wolff, Stewart MacKay, Wilmer Institute, Baltimore 5, Md., OP

Woodward, Paul Maxwell, Jr., 1515 First Ave., Middletown, Ohio, OP

Yourish, Norman B., 80 Jerusalem Ave., Hicksville, N. Y., OP

Zankan, Leo, 7232 Canby Ave., Reseda, Calif., OP

Ziskin, Daniel E., 6317 Wilshire Blvd., Los Angeles 48, Calif., OP

The following candidates have qualified for Fellowship in the Academy except in some single detail, such as certification of the signatures of sponsors. The deficiencies are expected to be made up before the Annual Business Meeting.

Almeda, Emmanuel M., Philippine General Hospital, Manila, Philippines, OP

Courtis, Baudilio, Parana 750, Buenos Aires, Argentina, OP

Clough, Howard Kenneth, Lankenau Medical Bldg., Philadelphia 31, Pa., OP

Foxworthy, Laurel Rae, 1511 Medical-Dental Bldg., Seattle 1, Wash., OP

Hernandez Gonzalo, Pedro A., Calle 8 #358, Vedado, Habana, Cuba, ALR

Kowal, Roland A., 505 South Oak Park Avenue, Oak Park, Ill.

Mackay, Murdo M., 321 Commercial St., Clifton Forge, Va., ALR

McNicholas, Patrick John, 288 Duckworth St., St. John's, Nfld., Canada, OP

Nalle, Ernest, Jr., 1312 Main, Lubbock, Texas, ALR

O'Mahony, John B., 327 N. Christina St., Sarnia, Ont., Canada, ALR

Skeehan, Raymond A., Jr., 130th Station Hospital, APO 403, New York, N. Y., OP

Tato, Juan Manuel, Azcuenaga 235, Buenos Aires, Cap., Argentina, ALR

Tonndorf, Juergen, Medical Research Center, Room 11, Iowa City, Iowa, ALR

PROGRAM IN BRIEF

Palmer House — Chicago

1958 Meeting

SATURDAY, OCTOBER 11

9:00 a.m.—Council Meeting—Private Dining Room 4

SUNDAY, OCTOBER 12

8:15 a.m. — Registration — Foyer to Grand Ballroom

9:00 a.m. — Ladies' Registration — Room 832

9:00 a.m.—Official Opening of Exhibits: Scientific—Private Dining Rooms 15, 16, 17; Club Foyer
Technical—Exhibition Hall and Red Lacquer Room

9:00 a.m.—Teachers' Sections: Ophthalmology and Otolaryngology Combined—Grand Ballroom

12:30 p.m.—Home Study Faculty Luncheon—Private Dining Room 9

4:30 p.m. — President's Reception — Grand Ballroom

8:00 p.m.—Special Scientific Program: Otosclerosis Study Group of the Committee on Conservation of Hearing—Grand Ballroom

8:00 p.m.—Special Scientific Program: American Orthoptic Council and American Association of Orthoptic Technicians—Williford Room, Conrad Hilton Hotel

MONDAY, OCTOBER 13

9:00 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

9:30 a.m.—Joint Scientific Session—Grand Ballroom

10:15 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

11:30 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ballroom

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

3:15 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

4:30 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

7:00 p.m.—Annual Banquet — Grand Ballroom

TUESDAY, OCTOBER 14

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ballroom

9:00 a.m.—ENT Instruction Courses—Seventh Floor and Crystal Room

9:00 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

10:15 a.m.—ENT Instruction Courses—Seventh Floor

10:15 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

11:30 a.m.—ENT Instruction Courses—Seventh Floor

11:30 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ballroom

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

3:15 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

4:30 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

5:30 p.m.—Dinner Meeting of International Association of Ophthalmological and Otolaryngological Secretaries—Private Dining Room 18

8:00 p.m.—Special Scientific Program: Committee on Plastic Surgery, Ophthalmology—Grand Ballroom

8:00 p.m.—Special Scientific Program: Committee on Plastic Surgery, Otolaryngology—Crystal Room

WEDNESDAY, OCTOBER 15

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ballroom

9:00 a.m.—ENT Instruction Courses—Seventh Floor and Crystal Room

10:15 a.m.—ENT Instruction Courses—Seventh Floor

11:30 a.m.—ENT Instruction Courses—Seventh Floor and Crystal Room

12:45 p.m.—Council Meeting Luncheon —Private Dining Room 5

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ballroom

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room
3:15 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room
4:30 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room
6:30 p.m.—Alumni Dinners

THURSDAY, OCTOBER 16

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures—Grand Ballroom
9:00 a.m.—ENT Instruction Courses—Seventh Floor and Crystal Room
10:15 a.m.—ENT Instruction Courses—Seventh Floor and Crystal Room
11:30 a.m.—ENT Instruction Courses—Seventh Floor and Crystal Room
12:30 p.m.—Faculty Buffet Luncheon—Private Dining Room 14
2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ballroom
2:00 p.m.—EYE Instruction Courses—Seventh Floor
3:15 p.m.—EYE Instruction Courses—Seventh Floor
4:30 p.m.—EYE Instruction Courses—Seventh Floor
5:30 p.m.—Business Meeting—Private Dining Room 18

FRIDAY, OCTOBER 17

8:45 a.m.—American Society of Ophthalmologic and Otolaryngologic Allergy—Private Dining Room 14

1858 1958

THE ILLINOIS DEPARTMENT OF PUBLIC WELFARE
joins in the
CENTENNIAL CELEBRATION
of the
ILLINOIS EYE AND EAR INFIRMARY
under the auspices of
THE ILLINOIS EYE AND EAR INFIRMARY ALUMNI
OPHTHALMOLOGY
and the
UNIVERSITY OF ILLINOIS OTOLARYNGOLOGIC
ALUMNI ASSOCIATION

Wednesday, October 15, 1958

Grand Ball Room
Palmer House

Cocktails at 6:00 P.M.
Dinner at 7:00 P.M.

$6.50 per plate

An outstanding program is planned.

Advance reservations should be made with the general chairman,
MR. LESTER R. GERBER, Superintendent
Illinois Eye and Ear Infirmary
904 West Adams Street, Chicago 7

All interested members of the ACADEMY, their ladies and guests are cordially invited, and are urged to make early reservations.

SCIENTIFIC SECTIONS

JOINT SESSION

Monday, October 13, 1958
9:30 a.m.

GRAND BALLROOM

Presiding Officer:
LEROY A. SCHALL, M.D.
President

Secretary:
EUGENE L. DERLACKI, M.D.

Call to Order.....The President

Announcements

Introduction of the Guest of Honor
Louis H. Clerf, M.D.
St. Petersburg, Florida

Address by the President
Science Knows No Frontiers

Address by the Guest of Honor
Retirement

Presentation of Honor Awards

RECIPIENTS OF HONOR AWARDS

Jerome W. Bettman
San Francisco

John E. Bordley
Baltimore

Guy L. Boyden
Portland, Oregon

Eugene L. Derlacki
Chicago

David D. DeWeese
Portland, Oregon

Edwin B. Dunphy
Boston

Arnold W. Forrest
White Plains, New York

Glen G. Gibson
Philadelphia

Dan M. Gordon
New York

Robison D. Harley
Atlantic City

Alexander A. Krieger
Pittsburgh

Mercer G. Lynch
New Orleans

Alfred E. Maumenee
Baltimore

P. Robb McDonald
Philadelphia

Joseph H. Ogura
St. Louis

Charles L. Schepens
Boston

Kinsey M. Simonton
Rochester, Minnesota

Byron Smith
New York

Isaac Tassman
Philadelphia

Frederick H. Theodore
New York

Everet H. Wood
Albuquerque

SYMPOSIUM:

THE CARE OF THE SURGICAL PATIENT

The Preoperative Evaluation of the Patient
H. Walter Jones, Jr., M.D.
Boston
BY INVITATION

Some Fallacies in Choice of Anesthetic Agents
William K. Hamilton, M.D.
Iowa City
BY INVITATION

Postoperative Surgical Care
Alton Ochsner, M.D.
New Orleans
BY INVITATION

AUTHORS OF PAPERS

All slides or films must be in the hands of the projection operator before the opening of each session.

Please be prepared to submit papers to the recorder immediately following presentation.

SECTION ON OTOLARYNGOLOGY

Monday, October 13, 1958

2:00 p.m.

GRAND BALLROOM

Presiding Officer:
LEROY A. SCHALL, M.D.
President

Secretary:
EUGENE L. DERLACKI, M.D.

THE LARYNX AND VOICE: LARYNGEAL PHYSIOLOGY UNDER DAILY STRESS

(A Motion Picture)

Hans von Leden, M.D.
and
Paul Moore, Ph.D.
BY INVITATION
Chicago

The second in a series of educational motion pictures on the larynx and voice, this film demonstrates the complex pattern of laryngeal function during everyday life. It presumes a knowledge of the fundamental anatomy and physiology of the larynx which was described in the first film, "The Function of the Normal Larynx."

To relate the varied physiologic changes to clinical observations, the larynx is first pictured in natural color with synchronized sound. The details of vibratory function are then brought out by ultra-slow motion pictures photographed at speeds up to 5,000 frames per second.

Several new and hitherto unreported phenomena are presented, including a biphasic pattern of laryngeal vibrations, the effect of intensity and air pressure upon the components of the vibratory cycle, and the significance of secondary or accessory waves. The slow-motion pictures also introduce new information on inspiratory sound production, on the motion of the loose mucous membrane in relation to the underlying musculature, and on the adjustments in different registers.

Illustrative graphs are used to explain the new concepts, and the adaptability of artificial models to the study of the vibrating mechanism is demonstrated.

2:30 p.m.

THE EFFECT OF LOUD NOISE ON TINNITUS

George F. Reed, M.D.
Boston

Discussers:

Hallowell Davis, M.D.
St. Louis

Aram Glorig, M.D.
Los Angeles

In an effort to shed more light on the problem of tinnitus, the author has investigated the effects of loud noise on subjective tinnitus. The work has been based on the proposal that tinnitus having its source in the inner ear usually originates in that area of the cochlea exhibiting decreased function; which area, in turn, corresponds to the frequencies of the sound experienced by the patient as tinnitus.

Animal experimentation has demonstrated the ability to produce selective destruction or damage of portions of the cochlea through exposure to high-intensity sound. This being so, it was further theorized that in humans, the application of high-intensity sound of a specific frequency could be used to produce further damage or destruction of the involved

areas of the cochlea with resultant lessening or loss of the tinnitus.

The author will describe in brief the methods used in the audiometric study of tinnitus, methods of noise traumatization, and results and observations made to date on approximately 100 patients.

3:00 p.m.

SURGICAL TREATMENT OF CARCINOMA OF THE EPIGLOTTIS BY LATERAL PHARYNGOTOMY

(Includes Motion Picture)

Max L. Som, M.D.
New York

Discusser:

Joseph H. Ogura, M.D.
St. Louis

Lateral pharyngotomy as a definitive surgical procedure for the extirpation of carcinoma of the epiglottis was originally described by Trotter and recently reported by Orton and Alonzo. After removal of the epiglottis, these authors routinely performed a temporary pharyngostomy with all its annoyances. In selected cases, wide excision with primary closure has been uniformly obtained by the following illustrated technique.

Limited lesions of the epiglottis, not involving the aryepiglottic folds and not extending distally beyond two centimeters above the anterior commissure, may be adequately removed by lateral pharyngotomy with preservation of laryngeal function. The epiglottic neoplasm can be excised in continuity with all the structures in the pre-epiglottic space. The hyoid bone and the upper half of the thyroid ala are removed for better exposure and wider excision.

When the carcinoma extended below the level of the hyoid bone, an elective neck dissection was performed in continuity with the epiglottectomy. The superior laryngeal nerve on the less involved side was isolated and preserved. By maintaining the head flexion with a plaster cast, primary closure was obtained without the use of the skin graft as employed by Ogura.

The decision to perform an epiglottectomy can best be made after adequate exposure by pharyngotomy. Permission must always be obtained to proceed with total laryngectomy and neck dissection in the event that the lesion is more extensive than had been previously determined.

The results after three to eight years' follow-up have been gratifying. Laryngeal function is good and local recurrence is rare. Cervical node metastases accounted for the few failures. It would appear that in selected cases an elective neck dissection in continuity with epiglottectomy and extirpation of the pre-epiglottic space is more curative than total laryngectomy.

3:35 p.m.

THE PATHOGENESIS OF ACUTE EXTERNAL OTITIS

Edley H. Jones, M.D.
Vicksburg, Mississippi
and
Tom D. Norman, M.D.
Jackson, Mississippi

BY INVITATION

Discussers:

James W. McLaurin, M.D.
Baton Rouge, Louisiana

Ben H. Senturia, M.D.
St. Louis

Our study on the pathogenesis of acute external otitis includes histories and clinical examination, with indicated clinical laboratory procedures, of 50 patients with histopathologic examination of tissue from fifty-two ears of these patients. While the majority of these are cases of acute external otitis, the series also includes cases of "itching ears," chronic external otitis, seborrheic dermatitis, contact dermatitis, eczematoid dermatitis, and suspected cases of atopic and neurogenic dermatitis. The clinical pictures in these various types have been correlated with the histopathologic findings. The purpose of the study is to determine the predisposing factors, the exciting factors and the clinical course of the various types of external otitis so that the diagnosis can be made promptly.

4:05 p.m.

THE SURGICAL RECONSTRUCTION OF POST-TRAUMATIC LARYNGEAL STENOSIS

(A Motion Picture)

Paul H. Holinger, M.D.
and
Paul W. Greeley, M.D.
BY INVITATION

Kenneth C. Johnston, M.D.
and
John W. Curtin, M.D.
BY INVITATION
Chicago

The successive steps in the surgical reconstruction of a larynx fractured in an automobile accident are illustrated in this film. Mirror and direct laryngoscopic views of the fractured larynx are compared with photographs of a normal larynx. This comparison emphasizes the degree of distortion and stenosis that results from lack of adequate early management of the injury. The principal portion of the film shows the surgical steps of the removal of scar and deformed cartilage to re-establish an airway. A stent graft is prepared and placed in the larynx. The film then demonstrates the endoscopic technique of removal of the foam rubber stent ten days later and the insertion of a polyethylene splint.

The subsequent course of healing of the graft is shown by mirror and direct laryngoscopic views four months and eight months after the external surgical operation. Final views compare the reconstructed larynx with the mirror views of the preoperative stenosis.

SECTION ON OPHTHALMOLOGY

Tuesday, October 14, 1958
9:00 a.m.

GRAND BALLROOM

Presiding Officer:
F. Bruce Fralick, M.D.
Vice-President

Secretary:
Kenneth L. Roper, M.D.

CYSTINOSIS

(A Clinicopathologic Case Report)

Levon K. Garron, M.D.
Oakland, California

Cystinosis (cystine storage disease) is a familial disease occurring in children in whom cystine is deposited in various organs and tissues of the body. It is probably related to Fanconi's syndrome. There are hereditary renal tubule defects which prevent the reabsorption of a variety of serum constituents and result in systemic deficiencies.

In this disease, the inability of the renal tubules to reabsorb phosphorus results in renal rickets or dwarfism. Alkaline phosphatase is re-released in the serum, and later glomerulonephritis and uremia supervene. Renal tubular defects which prevent the reabsorption of glucose and all of the amino acids may also be present. There is still no satisfactory explanation for the deposition of cystine all over the body, especially since the blood levels are not high.

The patient in the present case was admitted to the hospital at the age of 8 because of glomerulonephritis, renal rickets and diabetes insipidus. There was retardation of bone growth, enlarged liver and spleen, and photophobia. She was seen at the Eye Clinic, where the diagnosis of cystinosis was suggested by the appearance of the cornea and conjunctiva. These tissues were diffusely infiltrated by fine crystalline deposits; a biopsy of the conjunctiva revealed many rectangular and square crystals.

The patient died at the age of 9 of uremia complicated by pneumonia. Autopsy revealed deposits of cystine in the bone marrow, spleen, liver, lymph glands, eyes, adrenals and kidneys. There was chronic glomerulonephritis, osteomalacia and hyperplasia of the parathyroid glands.

9:10 a.m.

ANISEIKONIA

With Notes on the Jackson-Lancaster Controversy

(XV Jackson Memorial Lecture)

Arthur Linksz, M.D.
New York

9:50 a.m.

EVISCERATION THROUGH SCLERAL INCISION WITH INSERTION OF SPHERE

(A Motion Picture)

Wendell L. Hughes, M.D.
Hempstead, New York

The procedure depicted in this film is a modification of the Birge type of evisceration in which the cornea is preserved and the complete corneoscleral coat preserved with normal muscle attachments. A transverse incision through the conjunctiva is made over the insertion of the superior rectus tendon, and the muscle is removed from the eye. A transverse incision is then made through the sclera 3 or 4 mm. above the insertion of the superior rectus muscle. The contents of the eye are removed and the posterior surface of the cornea is curetted thoroughly. An implant is then placed inside the sclera through the opening above, and the scleral wound is closed with three mattress sutures, lapping the anterior layer over the posterior layer. The central suture is used to reattach the superior rectus muscle to the anterior layer, which aids in closure of the scleral wound. The conjunctival wound is then closed and the anterior surface of the cornea curetted thoroughly to reduce its sensitivity. A small sphere (12 to 14 mm.) is usually used. This type of operation preserves the depth of the fornices to allow good movement of the artificial eye.

10:10 a.m.

TUMORS OF THE LACRIMAL GLAND

Edmund B. Spaeth, M.D.
Philadelphia

Discussers:

John W. Henderson, M.D.
Ann Arbor, Michigan

Lorenz E. Zimmerman, M.D.
Washington, D. C.

Tumors of the lacrimal gland produce many symptoms similar to those occurring in other non-neoplastic orbital conditions. Because of this, differential diagnosis is of significant importance. There are differences in the types of neoplasm which appear in the lacrimal gland. These have varied mortality rates, and procedures quite different one from the other are required in their therapy. Many ophthalmologists are convinced that the surgical therapy of various lacrimal gland neoplasms, as frequently carried out at the present, is quite inadequate when considering the high mortality rates from malignant lesions of the lacrimal gland.

10:40 a.m.

A LIVING CYSTICERCUS WITH ACTIVE MOVEMENTS IN THE ANTERIOR CHAMBER

(A Motion Picture)

Teodulo Manuel Agundis, M.D.
Mexico, D. F.
BY INVITATION

10:50 a.m.

FACTORS IN REDUCING OCULAR PRESSURE PRIOR TO INTRAOCULAR SURGICAL PROCEDURES

William G. Everett, M.D.
and
E. Kenneth Vey, M.D.
BY INVITATION

Cornelius Y. Veenis, M.D.
BY INVITATION
Pittsburgh

Discussers:

Harold Gifford, M.D.
Omaha

Ralph E. Kirsch, M.D
Miami, Florida

The purpose of this study was to analyze the factors causing a reduction of intraocular pressure in ophthalmic surgical procedures.

A control group consisted of one hundred patients who had had a routine cataract extraction performed under local anesthesia, using 1.5 per cent monocaine with five turbidity reducing units (TRU) of Wydase per cubic centimeter. The intraocular tension of both eyes was determined on admission, after preoperative sedation, after retrobulbar injection of local anesthesia, and following five minutes of digital pressure. The degree of akinesia following anesthesia was measured.

Patients with glaucoma comprised a second group. In addition, gonioscopy and tonographic measurements were performed. The reduction of intraocular pressure in these glaucoma patients was compared to that in members of the control group. The reduction of intraocular tension after Pentothal anesthesia was also compared to that in the control series.

The results of this study showed a definite fall in intraocular tension after preoperative sedation; however, this fall is less predictable than the fall in pressure after retrobulbar injection or digital pressure. There is a definite, but less significant, reduction of intraocular pressure after Pentothal anesthesia. Finally, the fall in intraocular pressure following digital pressure in glaucoma cases seems to be proportionate to the facility of outflow.

Other observations will be reported.

11:20 a.m.

TUMORS AND MAJOR INJURIES OF THE LOWER LID: FUNCTIONAL AND STRUCTURAL REPAIR

(A Motion Picture)

Alston Callahan, M.D.
Birmingham, Alabama

A group of reconstructive procedures, designed to cover most problems of lower lid reconstruction, is shown in this film. In the first patient, a carcinoma in the middle of the lower lid was destroyed by roentgen irradiation. Severe ectropion resulted, so that reconstruction of the anterior structure of the lower lid was required. Contraction of the free skin graft from abnormal vertical lid tension was prevented by an intermarginal lid adhesion.

In the second case, reconstruction of the medial half of the lower lid with the Hughes procedure, using the lower half of the posterior surface of the upper lid and a free skin graft, is shown.

In the third case, the lateral half of the lower lid is reconstructed with a pedicle transposed from the upper lid.

In the last case, the left eye and lower lid were lost from a bullet wound. Reconstruction with pedicles of skin from the upper lid and from the zygomatic region is shown.

11:40 a.m.

THE TREATMENT OF IRIS MELANOMA

Algernon B. Reese, M.D.
New York
and
Gilbert W. Cleasby, M.D.
San Francisco
BY INVITATION

Discusser:

Benjamin Rones, M.D.
Washington, D. C.

Analysis of 157 cases of malignant melanoma of the iris seen at the New York Eye Institute reveals that these tumors fall into three definite groups from the standpoint of treatment. These groups consist of those lesions best handled by observation, by local excision, and by enucleation, respectively. The indications for each approach will be discussed, the technique for local excision presented, and the over-all prognosis given.

SECTION ON OTOLARYNGOLOGY

Tuesday, October 14, 1958

2:00 p.m.

GRAND BALLROOM

Presiding Officer:
DAVID D. DEWEESE, M.D.
Vice-President

Secretary:
EUGENE L. DERLACKI, M.D.

AN APPROVED TECHNIQUE FOR PURE TONE, AIR CONDUCTION AUDIOMETRY

(A Motion Picture)

Research Center
Subcommittee on Noise

Aram Glorig, M.D.
Los Angeles

This film is designed to present an approved technique for determining the auditory threshold by air conduction with pure tones.

Our experience has demonstrated that industrial audiometry, school audiometry, and in many cases, the audiometry done in doctors' offices, leave much to be desired. It is hoped that this film will assist in promoting a standard technique.

The prospective audiometrist is taken through the audiometric procedure step by step. The important steps and the many pitfalls are emphasized by live photography, animation and narration. The importance of good audiometry has increased immeasurably. We believe the film will go a long way toward improving pure tone air conduction threshold audiometry.

The film was prepared by the Research Center, Subcommittee on Noise of the Committee on Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology. It may be obtained on a loan basis for transportation charges or bought for cost by writing to the Research Center, Subcommittee on Noise, 111 North Bonnie Brae Street, Los Angeles 26, California.

2:20 p.m.

DELAYED SIDETONE TEST TO DETECT UNIAURAL NONORGANIC DEAFNESS: ITS CLINICAL APPLICATION

Edward W. Gibbons
BY INVITATION

Richard A. Winchester, Ph.D.
BY INVITATION
Los Angeles

Discussers:

Raymond Carhart, Ph.D.
Evanston, Illinois
BY INVITATION

Scott N. Reger, Ph.D.
Iowa City

Since January 1956, the Veterans Administration Audiology and Speech Correction Clinic at Los Angeles, California, has engaged in a clinical investigation of 550 veterans initially presenting uniaural deafness. For the purposes of this study, uniaural deafness was defined as any auditory deficit showing one ear falling within the limits of normal hearing with the contralateral ear showing a relative difference in hearing level greater than 30 decibels.

The first result of this investigation was a description of a delayed sidetone test designed to detect uniaural nonorganic deafness, which appeared in the Archives of Otolaryngology for July 1957. Since July 1957 this testing procedure has also been applied experimentally to the problem of demonstrating the existence of bonafide organic uniaural deafness.

The present report concerns revisions made in the test, together with results of its clinical application in a group of selected cases which presented special audiometric problems. The results of this experimental application of the testing procedure have demonstrated that the revisions of the original test were justi-

fied, and that, because of the test's effectiveness, it should be included in the standard battery of tests designed to detect uniaural nonorganic deafness.

2:55 p.m.

SURGICAL REPAIR OF TRAUMATIC INTERRUPTION OF THE OSSICULAR CHAIN

(A Motion Picture)

George E. Shambaugh, Jr., M.D.
Eugene L. Derlacki, M.D.
Wiley H. Harrison, M.D.
Chicago

Experimental studies have indicated that interruption of the ossicular chain with an intact tympanic membrane causes a particularly severe conductive hearing impairment. Four cases have been encountered, three resulting from a previous simple mastoidectomy and one from head trauma. In all there was a severe conductive loss with a large air-bone gap. Surgical repair by restoring the continuity between the tympanic membrane and the stapes resulted in a substantial hearing improvement with restoration of hearing that was close to normal in some cases.

3:30 p.m.

PATHOLOGY OF CRICOARYTENOID ARTHRITIS

William W. Montgomery, M.D.
Boston

Discussers:

LeRoy A. Schall, M.D.
Boston

Mercer G. Lynch, M.D.
New Orleans

The etiology, signs and symptoms and laryngeal picture of cricoarytenoid arthritis are briefly discussed. These are based on the findings in seventeen cases.

Two case histories of cricoarytenoid arthritis are presented; one patient died of other disease and the second as a direct result of cricoarytenoid arthritis. The pathologic changes in these cases are shown and compared with normal cricoarytenoid joints. Complete fibrous ankylosis is not shown but severe arthritic changes are quite apparent.

4:00 p.m.

ELECTIVE RHINOPLASTY

(A Motion Picture)

Irving B. Goldman, M.D.
New York

In this film the surgical technique and procedures of a rhinoplasty are shown in detail in an actual clinical and operative presentation. Since the film is to be used as a teaching aid, many of the operative steps are demonstrated on a model that exactly reproduces the osseous upper cartilaginous and lower cartilaginous vaults. Special attention is given to the precise management and function of instruments, handling of tissues and avoidance of operative and postoperative reactions. The prophylactic use of corticosteroids is emphasized. The action is climaxed by the appearance of the patient, showing rapid return to relative normalcy of the surgical nose.

SECTION ON OPHTHALMOLOGY

Wednesday, October 15, 1958
9:00 a.m.

GRAND BALLROOM

Presiding Officer:
PHILIP M. LEWIS, M.D.
Vice-President

Secretary:
KENNETH L. ROPER, M.D.

INVOLVEMENT OF THE EYES IN PERIARTERITIS NODOSA

(A Clinicopathologic Case Report)

Frederick C. Blodi, M.D.
Iowa City

Paul B. Sullivan, M.D.
Hanover, New Hampshire

A 64-year-old white female patient suffered from cardiovascular hypertension for three years. The blood pressure dropped under treatment from 190/110 to 160/100. Nonprotein nitrogen rose to 159, and albuminuria with hematuria occurred. Numerous hemorrhages in skin, gastrointestinal tract and retinas appeared. Biopsy of the deltoid muscle revealed polyarteritis nodosa. The diagnosis was confirmed at autopsy.

Both eyes showed severe hypertensive retinopathy with retinal detachment. Active foci of choroiditis were present. This case shows that detachment of the retina in periarteritis nodosa is usually due to the hypertensive retinopathy and not to arteritis of the choroidal vessels. The finding of a nongranulomatous uveitis in this collagen disease is of great theoretical interest and may be more frequent than assumed.

9:10 a.m.

GLAUCOMA SURGERY

(A Motion Picture)

Enrique Malbran, M.D.
Buenos Aires, Argentina
BY INVITATION

The film includes the author's pathogenic and clinical classification of primary glaucomas. The surgical procedure to be used in each case is chosen according to this classification.

The technique of peripheric iridectomy with retention of the anterior chamber is shown. This operation is especially adapted for use as a prophylactic method, or as first choice for preglaucoma and interval narrow closed angle in which the intraocular pressure and the outflow (tonographic "C") remains normal after the crisis. Preoperative and postoperative goniophotographs of these types of glaucoma are presented.

A new technique for filtering operation is proposed as particularly useful for chronic closed angle glaucoma, for the mixed types, and for narrow open angle glaucomas. The operation is called filtering peripheric iridectomy. The main purpose of the new procedure is to obtain a definite filtering scar by the retraction of both lips of the wound when diathermic coagulation is applied. Surgical approach is by a previous "ab externo" incision. This type of operation is safer and simpler to perform than classical filtering procedures. With this new technique a round pupil is maintained and the whole anatomy of the eye is conserved. Preoperative and postoperative goniophotographs and external views are presented in the film.

Finally, the results obtained by this procedure for the first 50 patients on whom it was done are shown. Approximately 82 per cent of the results were good (normal intraocular pressure and improvement of the tonographic "C").

9:30 a.m.

SYMPOSIUM: OCULAR EFFECTS OF IONIZING RADIATION AND RADIOTHERAPY

BASIC CONCEPTS

Orientation

David G. Cogan, M.D.
Boston

Physical and Biological Effects of Ionizing Radiation

Titus C. Evans, Ph.D.
Iowa City
BY INVITATION

Some Experimental Ocular Lesions Resulting from Ionizing Radiation

Ludwig von Sallmann, M.D.
Bethesda, Maryland

RADIOTHERAPY

Radiotherapy of Ocular Adnexa

Milford W. Schulz, M.D.
Boston

Radiotherapy of Ocular Lesions by X-rays and Gamma Rays

Arnold W. Forrest, M.D.
White Plains, New York

Radiotherapy of Ocular Lesions by Beta Particles

James E. McDonald, M.D.
Oak Park, Illinois

Fred M. Wilson, M.D.
Indianapolis

Radiotherapy Compared to Other Forms of Ocular Therapy

William F. Hughes, Jr., M.D.
Chicago

SECTION ON OTOLARYNGOLOGY

Wednesday, October 15, 1958

2:00 p.m.

GRAND BALLROOM

Presiding Officer:
LeRoy A. Schall, M.D.
President

Secretary:
Eugene L. Derlacki, M.D.

PRESENTATION OF NEW INSTRUMENTS

2:30 p.m.

FULL FACE REPLACEMENT IN A ONE-STAGE OPERATION FOR THE TREATMENT OF EXTENSIVE CANCER

(A Motion Picture)

John J. Conley, M.D.
New York

The development of multiple primary squamous cell cancers in the skin of the face, or within ulcers occurring as a result of severe radiation dermatitis, requires aggressive and definitive therapy. A one-stage surgical procedure in which all of the involved skin is excised and replaced with rotated pedicle flaps from the neck offers an excellent expectation of cure of the disease and esthetic improvement.

2:50 p.m.

BASIC PRINCIPLES USED IN THE DEVELOPMENT OF MODERN TEMPORAL BONE SURGERY TECHNIQUE

(XIV Wherry Memorial Lecture)

Julius Lempert, M.D.
New York

3:50 p.m.

SURGICAL MANAGEMENT OF ORAL CANCER

Maurice F. Snitman, M.D.
Joseph G. Schoolman, M.D.
Arthur Loewy, M.D.
and
Mario D. Mansueto, M.D.
BY INVITATION
Chicago

The employment of surgery in the management of intraoral lesions during the past decade represents an advance of great significance in the therapy of neoplastic disease about the head and neck. The general application of some form of irradiation (radium or x-ray) in the past not only resulted in a very poor survival rate, but in an unpredictable and uncontrolled morbidity in that there was considerable pain and untoward sequelae.

Improvement of survival rates and morbidity by surgical measures is especially to be noted in resectable lesions of the tongue, floor of mouth, hard palate and gingiva. It is most gratifying that the resulting deformity interferes but little with normal physiologic processes.

The surgical approach takes into consideration the management of the primary lesions and the cervical nodes. Floor of the mouth and lower gingival lesions are treated by removal of the primary lesion, with what is generally considered to be radical neck dissection of the same side, but which we term, by preference, complete or adequate. The neck dissection is carried out under two conditions: (1) in the absence of palpable nodes, when primary lesion is advanced and fixed; and (2) when palpable nodes are present but considered operable.

Unilateral lesions of the body of the tongue without palpable nodes are not treated by the composite (surgery in continuity) procedure because of the possibility of crossed metastases.

4:10 p.m.

RADICAL SURGICAL APPROACHES IN CANCER OF THE ORAL CAVITY

Eugene L. Grandon, M.D.
Iowa City
BY INVITATION

Uncontrolled carcinoma of the floor of the mouth is a painfully disabling, miserable, fatal disease.

In recent years carcinoma of the oral cavity has been managed largely by radiation therapy even when the disease is adjacent to bone but without radiological evidence of bone destruction. There are few centers in this country with a wide experience in effectively treating carcinoma of the floor of the mouth with radiotherapy. In extensive lesions, palliation is more commonly undertaken.

Surgical procedures still provide one of the most effective methods in treating this disease. During the past several years we have employed a variety of surgical procedures to cure this disease, keeping in mind an attempt to maintain physiological function of the tongue as well as understandable speech.

Discussers of Preceding Two Papers:

John J. Conley, M.D.
New York

Donald A. Corgill, M.D.
Dallas

SECTION ON OPHTHALMOLOGY

Thursday, October 16, 1958

9:00 a.m.

GRAND BALLROOM

Presiding Officer:
F. Bruce Fralick, M.D.
Vice-President

Secretary:
Kenneth L. Roper, M.D.

ENDOGENOUS INTRAOCULAR FUNGUS INFECTION

(A Clinicopathologic Case Report)

Robison D. Harley, M.D.
Jay E. Mishler, M.D.
Atlantic City

Endogenous intraocular fungus infection is a relatively rare diagnosis. Fourteen cases have been previously reported. An additional case resembling Aspergillus infection is described. By including the present case, Aspergillus was the causative organism in seven of the fifteen reported cases.

The unusual feature of the case here reported is the apparent deleterious effect of systemic corticosteroid therapy on vitreous abscess formation due to Aspergillus.

9:10 a.m.

PRESENTATION OF NEW INSTRUMENTS

9:25 a.m.

EXTRACTION OF CATARACT IN THE YOUNG ADULT: CAPSULOTOMY AND IRRIGATION WITH FUCHS' SYRINGE

(A Motion Picture)

Louis Daily, M.D.
Houston, Texas

Assembly and operation of Fuchs' double-lumened cannula and syringe are demonstrated. Extracapsular extraction of lamellar cataracts from both eyes of a young adult aided by simultaneous irrigation and aspiration with Fuchs' syringe is shown. Fluid is ejected from the bore of the syringe below the plunger, and fluid and lenticular material are aspirated into the syringe above the plunger by way of the double-lumened cannula. When Fuchs' syringe is used, the corneal incision may be smaller than that usually required for extracapsular extraction of cataract.

9:35 a.m.

THE ELECTRORETINOGRAM IN HEREDITARY VISUAL CELL DEGENERATION

Albert D. Ruedemann, Jr., M.D.
Detroit
and
Werner K. Noell, M.D.
Buffalo

BY INVITATION

Discusser:

Jerry H. Jacobson, M.D.
New York

Electroretinograms (ERG) from forty cases of retinitis pigmentosa will be discussed and compared with the electroretinograms obtained in an inbred strain of mice (C3H) which is affected with hereditary visual cell degeneration. Measuring and recording techniques were the same in both studies. In retinitis pigmentosa there were no electric variations in the retina when no peripheral vision had remained; otherwise the variations were present but the ERG was abnormal in configuration and amplitude. Visual deficiencies in cases in which it was possible to obtain an ERG had not been noted by the patient prior to the age of 25. The vessels were not always markedly attenuated nor were the discs particularly waxy. Some of the patients in these cases had a family history of retinitis pigmentosa.

In the mice the disease was characterized by death of the visual cells following a few days after the visual cells had postnatally differentiated to a functional state. As visual cell differentiation progressed from central regions toward the periphery, the area of visual cell death increased correspondingly. The electric vibrations in the retina became extinct when the visual cells at the ora serrata degenerated and no functional visual cells remained. The vibrations developed in these mice as early as in normal control strains, but the ERG always had an abnormal configuration. The C3H ERG and the abnormal ERG in retinitis pigmentosa showed a similar increase in the latency of the a-wave. In the mouse, the a-wave amplitude was also preferentially reduced, whereas in retinitis pigmentosa it was present only in some of the cases. A similar increase in a-wave latency resulted in rabbits from iodate poisoning in conjunction with outer and inner limb degeneration. The pathology of retinitis pigmentosa will be reviewed in relation to these and other findings in animals.

10:05 a.m.

EFFECT OF ELECTRICAL STIMULATION OF THE CILIARY GANGLION ON THE ANTERIOR SEGMENT OF THE CAT'S EYE

(A Motion Picture)

Mansour F. Armaly, M.D.
Iowa City

BY INVITATION

In the cat, removal of parasympathetic tone reduces, and stimulation increases, the facility of outflow.

The fibrillar meshwork of the angle is arranged in sets of fibers lateral to each other, with fibers of one set overlapping interfibrillary spaces of adjoining layers. This arrangement makes the degree of separation of these layers as well as the spatial relationship of the fibers of critical importance in determining magnitude of its resistance to outflow.

The connection of this meshwork to the meridional and circular fibers of the ciliary muscle makes contraction or relaxation of this muscle alter the tension in these fibers (hence their resistance to compression by the intraocular pressure) and their spatial arrangement.

By direct visualization, contraction of the ciliary muscle is seen to produce recession and medial displacement of its attachment to the meshwork and iris, with increase in angle depth, tension and length of the visible fibers, and separation of the laterally placed sets of fibers. The central six millimeters of the anterior surface of the lens bulges forward, covered by the constricting iris leaf.

10:10 a.m.

INDICATIONS AND LIMITATIONS OF LIGHT-COAGULATION OF RETINAL LESIONS

Doz. G. Meyer-Schwickerath
Bonn, West Germany
BY INVITATION

10:30 a.m.

CORNEAL "MUSHROOM" GRAFT

(A Motion Picture)

Frederick W. Stocker, M.D.
Durham, North Carolina

Prior to the showing of the motion picture, a few still slides will be presented in order to make it easier to follow the various steps of the technique.

The outline of the lamellar part is made on the donor cornea with a 10 mm. trephine. A few marks are made on the surface of the cornea with calipers, indicating the distance of the central perforating part of the graft from the margin of the lamellar part. The lamellar part then is dissected up to the central area, where the perforating part has been marked. The whole cornea is then excised with cataract knife and scissors and placed with endothelial side up on the paraffin block in which an indentation corresponding to the corneal curvature has been made. A 5 mm. trephine then is applied in the center on the endothelial side and the inner part of the perforating stem cut until the lamellar portion is reached. The excision of the surplus tissue peripheral to the central perforating part is completed with scissors. A graft the shape of a mushroom with a large lamellar and a small central perforating part is obtained.

An outline of the lamellar part to be removed from the recipient's eye is made with the 10 mm. trephine that was used for the donor. The superficial layers of the cornea within that outline are then removed. The location of the central perforation in which the perforating part of the graft is to be placed is marked with calipers, as was done with the donor cornea. The 5 mm. trephine is then applied in that central area, and a through-and-through cut over the cornea is made. After the plug is removed, the graft is immediately put into place. The peripheral lamellar part is sutured to the recipient's cornea with 6-0 silk sutures. At the completion of the operation, a clear central perforating part can be seen, whereas the peripheral part of the cornea still appears cloudy, because some of the scar tissue, which comprises the deeper layers of the cornea, is still present. At the end, the patient's eye is shown two months after the operation.

This technique is recommended for the type of case which is known to respond poorly to perforating grafts, such as heavily vascularized scars after chemical burns, vascularized unsuccessful grafts, and severe bullous keratopathy after cataract extraction.

10:45 a.m.

OPTICAL AIDS FOR LOW VISUAL ACUITY

Gerald Fonda, M.D.
Short Hills, New Jersey

Daniel Snydacker, M.D.
Chicago

The use of optical aids for patients with low visual acuity has increased greatly in the past few years; yet, there remain many patients who might be benefited from such devices.

Fitting these aids is neither complicated nor difficult. Various types of aids will be described and criteria for their successful use listed.

It will be emphasized that these aids can be prescribed by any practitioner so that the patient does not have to be referred to a special clinic.

11:15 a.m.

PAPILLEDEMA CAUSED BY IRON DEFICIENCY ANEMIA

Marvin J. Lubeck, M.D.
Ann Arbor, Michigan
BY INVITATION

Discusser:

Ernest Beutler, M.D.
Chicago
BY INVITATION

A case of bilateral papilledema in a 14-year-old girl with iron deficiency anemia is presented. The papilledema was the result of the anemia and was caused by increased intracranial pressure. The anemia promptly responded to iron therapy with simultaneous clearing of the papilledema. There are eleven cases in the literature that are essentially the same, the most recent in 1936. Most were considered as cases of chlorosis with the papilledema an ocular complication. (Chlorosis was characterized by a microcytic, hypochromic anemia of obscure origin, occurring primarily in teen-age girls. It is considered an extinct disease.)

A tentative diagnosis of brain tumor was made on the basis of a history of headache, a suspicious skull x-ray finding, and bilateral papilledema. Eye examination revealed a visual acuity of 20/30 in each eye and bilateral papilledema with hemorrhages, exudates, and macular star figure. Visual fields showed general constriction and enlargement of the blind spots. The hemoglobin was 4.8 grams. The lumbar puncture opening pressure was 280 millimeters of water. Thorough study of the patient revealed no cause for the anemia. Iron therapy resulted in relief from symptoms and disappearance of the papilledema with the rise of the hemoglobin level.

Lumbar puncture had not been done in any of the previously reported cases. The elevated pressure in this case and a prior report that patients with chlorosis, although without papilledema, frequently had high lumbar puncture pressures suggest that the papilledema was due to increased intracranial pressure. The reversibility of the process and reports of autopsies on patients with papilledema due to systemic disease point to edema of the brain as the cause of the increased intracranial pressure.

11:45 a.m.

THE DIAGNOSTIC VALUE OF FLICKER PERIMETRY IN CHRONIC SIMPLE GLAUCOMA

Charles J. Campbell, M.D.
BY INVITATION
M. Catherine Rittler
BY INVITATION
New York

Discusser:

Paul W. Miles, M.D.
St. Louis

Flicker perimetry has been carried out on a clinical basis using a gray tangent screen and a stroboscopic lamp mounted on a wand. Determinations were made of the maximum frequency of stimulation at which flicker was seen for selected points in the visual field. Testing a large number of normal observers provided a range of values for these points. In a similar manner, a group of patients with chronic simple glaucoma was studied, and their data compared quantitatively to those obtained for the normal series. The glaucoma patients were also studied with conventional visual fields, and these findings were related to the flicker abnormalities previously determined.

It is concluded that flicker perimetry can be a sensitive technique for detecting small functional changes when the testing conditions are carefully standardized. In many cases this test is more rapidly performed and is less fatiguing to the patient than conventional visual fields.

SECTION ON OTOLARYNGOLOGY

Thursday, October 16, 1958
2:00 p.m.

GRAND BALLROOM

Presiding Officer:
DAVID D. DEWEESE, M.D.
Vice-President

Secretary:
EUGENE L. DERLACKI, M.D.

CYTOLOGIC DIAGNOSIS IN OTOLARYNGOLOGY

(Sponsored by The American Society of Ophthalmologic and Otolaryngologic Allergy)

William T. K. Bryan, M.D.
and
Marian P. Bryan
BY INVITATION
St. Louis

Discussers:

French K. Hansel, M.D.
St. Louis

Joseph L. Goldman, M.D.
New York

The distinct cellular patterns which many diseases present are valuable for diagnostic purposes. With improvement in techniques, more complete and careful observations of all cellular elements present in smears can be seen and evaluated. Useful cytologic patterns have been established for the following conditions: acute cold, nasal allergy, bacterial rhinosinusitis, measles, and tumors, especially those that are malignant. In ear secretions the differential diagnosis of acute and chronic otitis media and mastoiditis as well as cholesteatoma and external otitis is useful. In reporting a smear, an evaluation is made of all the elements found so that the report may well be called a "cytogram." The material is obtained by direct wiping of the area under examination. This is actually a painless nontraumatic biopsy. When tumor cells are found, tissue study is indicated.

Of especial interest in the cytograms are the degenerating epithelial cells of acute upper respiratory infections, the goblet cells and mast cells in nasal allergy, the giant cells of measles, herpes and chickenpox, and the phagocytes of mastoiditis. These features and those of malignant cells will be illustrated.

2:40 p.m.

THE SURGICAL CORRECTION OF APHAGIA FOLLOWING BULBAR POLIOMYELITIS

(A Motion Picture)

Benjamin Bofenkamp, M.D.
Minneapolis

The purpose of this film is to demonstrate the author's technique for restoration of ability to swallow following complete inability resulting from bulbar poliomyelitis.

The film first shows a patient preoperatively feeding herself through a gastrotomy, indicating the marked disability the aphagia produces. Then a detailed anatomy of the hypopharynx with particular reference to the cricopharyngeus muscle is presented. The surgical technique is diagrammatically outlined, and an actual surgical case is presented. Roentgenograms of the patient's swallowing mechanism both preoperatively and postoperatively are also shown. The final portion of the film demonstrates a patient eating normally six weeks postoperatively as contrasted with her preoperative nasal gastric tube feeding.

3:00 p.m.

SURGICAL TECHNIQUE FOR SUBTOTAL TRACHEAL RESECTION

William R. Waddell, M.D.
Boston
BY INVITATION

Discusser:

Jerome R. Head, M.D.
Chicago
BY INVITATION

3:30 p.m.

INDICATIONS AND SURGICAL MANAGEMENT OF CHRONIC EAR DISEASE

Howard P. House, M.D.
William F. House, M.D.
Los Angeles

Discusser:

Wesley E. Compere, Jr., M.D.
Los Angeles

There are definite indications for the conventional modified and radical mastoid operations in cases of chronic ear disease. Likewise, there are definite in-

dications for myringoplasty and tympanoplasty which are performed in an attempt to restore middle ear function.

The indications for these various procedures, along with a general description of surgical management, will be presented.

4:10 p.m.

PRECIS OF MOBILIZATION TECHNIQUES

(A Motion Picture)

Edmund P. Fowler, Jr., M.D.
Milos Basek, M.D.
and
Vladimir Epanchin, M.D.
BY INVITATION
New York

A compendium of various methods of mobilizing the ankylosed stapes footplate showing favorite tools and basic techniques of several American operators in 1958. Among them are Altmann, Bellucci, Goodhill, House, Rosen, Schuknecht, Shambaugh, Shea and the present authors. The pictures are of these men working on partially dissected cadaver material with occasionally actual, but usually simulated (dental cement), otosclerotic foci. The manipulations are shown for the most part simultaneously on the middle ear and the vestibular aspects of the footplate. Penetration into the vestibule with needles, picks, harpoons, chisels, drills, and hammers is shown as well as the effect of stapes head and crural manipulations on the footplate. Apparently many operators are achieving much the same end, namely footplate perforation, displacement or fracture in spite of superficial differences in theory or technique.

SPECIAL SCIENTIFIC PROGRAMS

These Special Programs are open meetings. All members and guests of the Academy are welcome to attend.

TEACHERS' SECTIONS— OPHTHALMOLOGY and OTOLARYNGOLOGY

Grand Ballroom

Sunday, 9:00 a.m.

JAMES H. MAXWELL, M.D.
A. D. RUEDEMANN, M.D.
Co-Chairmen

ARE WE GOOD TEACHERS?

This presentation offers practical considerations and demonstrations in the elements of which good pedagogy is formed.

Introductory Remarks

FRANCIS L. LEDERER, M.D.
Chicago

What Is Involved in the Learning Process?

LAWRENCE K. FRANK
Belmont, Massachusetts
BY INVITATION

What Are the Factors in Effective Pedagogy?

RAY N. LOWE, ED.D.
Eugene, Oregon
BY INVITATION

What Are Effective Teaching Tools and How Can They Be Used Effectively?

RICHARD B. LEWIS, ED.D.
San Jose, California
BY INVITATION

It is estimated that this presentation will stimulate further exploration into the many facets of effective pedagogy.

Summation for Otolaryngology

ALBERT C. FURSTENBERG, M.D.
Ann Arbor, Michigan

OTOSCLEROSIS STUDY GROUP and COMMITTEE ON CONSERVATION OF HEARING

Grand Ballroom

Sunday, 8:00 p.m.

LAWRENCE R. BOIES, M.D.
DEAN M. LIERLE, M.D.
Co-Chairmen

Observations, Facts and Recommendations Derived from a Mental Health Project for the Deaf

JOHN D. RAINER, M.D.

FRANZ J. KALLMANN, M.D.
New York
BY INVITATION

Histopathologic Observations Following Fenestration and Stapes Mobilization

JOHN R. LINDSAY, M.D.
Chicago

Antibiotics and the Inner Ear

JOSEPH E. HAWKINS, JR., PH.D.
New York
BY INVITATION

Discusser:

GEORGE E. SHAMBAUGH, JR., M.D.
Chicago

AMERICAN ORTHOPTIC COUNCIL

and

AMERICAN ASSOCIATION OF ORTHOPTIC TECHNICIANS

Williford Room

Conrad Hilton Hotel

Sunday, 8:00 p.m.

EDMOND L. COOPER, M.D.
Secretary

SYMPOSIUM: OFFICE ORTHOPTICS BY THE OPHTHALMOLOGIST

Introduction and Case Selection
WEBB P. CHAMBERLAIN, JR., M.D.
Cleveland

Orthoptics: An Aid in Diagnosis
FLORENCE M. MACLEAN,
Washington, D. C.

The Worth Amblyoscope and the Stereoscope in Action
JACK P. COWEN, M.D.
Chicago

Office Therapy: Limitations and Possibilities
FRANCES FOWLER
Los Angeles

COMMITTEE ON RECONSTRUCTIVE PLASTIC SURGERY: OPHTHALMOLOGY

Grand Ballroom

Tuesday, 8:00 p.m.

WENDELL L. HUGHES, M.D.
Chairman

SYMPOSIUM: PTOSIS COMPLICATIONS

CHARLES E. ILIFF, M.D.
Moderator
Baltimore

Introduction

a. Anatomy

b. Physiology

F. BRUCE FRALICK, M.D.
Ann Arbor, Michigan

Discussion

RAYNOLD N. BERKE, M.D.
Hackensack, New Jersey

JOHN S. CRAWFORD, M.D.
Toronto, Ont., Canada

ROBERT A. SCHIMEK, M.D.
New Orleans

COMMITTEE ON RECONSTRUCTIVE PLASTIC SURGERY: OTOLARYNGOLOGY

Crystal Room

Tuesday, 8:00 p.m.

JOHN MARQUIS CONVERSE, M.D.
Chairman

The Surgical Correction of Nasal Skeletal Defects
EDGAR M. HOLMES, M.D.
Boston

General Discussion

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

Private Dining Room 14

Friday, 8:45 a.m.

JOSEPH W. HAMPSEY, M.D.
President

Introductory Remarks by Dr. Hampsey

9:00 a.m.

Natural Resistance to Infection: The Properdin System

IRWIN H. LEPOW, PH.D.
Cleveland

10:00 a.m.

Toxic Bacterial Antigens: Their Mechanism of Action

A. I. BRAUDE, M.D.
Pittsburgh

11:05 a.m.

Cellular Physiology

(A New Motion Picture)

THOMAS F. DOUGHERTY, PH.D.
Salt Lake City

11:35 a.m.

The Significance of the Mast Cell in Nasal Secretion

WILLIAM T. K. BRYAN, M.D.
and
MARIAN P. BRYAN
St. Louis

12:05 p.m.

Sympathetic Ophthalmia as an Allergic Phenomenon

FREDERICK C. BLODI, M.D.
Iowa City

12:35 p.m.

Panel Discussion

FRENCH K. HANSEL, M.D.
Moderator
St. Louis

DR. LEPOW; DR. BRAUDE;
DR. DOUGHERTY; DR. BLODI

1:05 p.m.

Business Meeting

SCIENTIFIC EXHIBITS

KENNETH L. ROPER, M.D., *Chairman*

EUGENE L. DERLACKI, M.D., *Co-Chairman*

CHICAGO, ILLINOIS

LOCATION:

Registration Area, 4th Floor

Home Study Courses, American Academy of Ophthalmology and Otolaryngology

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, represented by Daniel Snydacker, M.D., Secretary for Home Study Courses, Chicago, and Ben H. Senturia, M.D., Associate Secretary in charge of Otolaryngology, St. Louis

This exhibit will show the purposes, scope and organization of the Home Study Courses.

American Board of Opticianry and Educational Foundation in Ophthalmic Optics

CHARLES SHEARD, Ph.D., Secretary of the American Board of Opticianry, Rochester, Minnesota, and Howard Quackenbush, Chairman, Exhibit Committee, Columbus, Ohio

The exhibit will be concerned with the purposes and functions of the Board in matters of collegiate education, both theoretical and practical, in opticianry and the certification, by examination only, of graduates of schools and opticians in practice; also, the work of its Educational Foundation in matters of scholarships and other awards of merit.

LOCATION:

Club Floor

Exhibit S-1

Speech Rehabilitation—One Easter Seal Service

THE NATIONAL SOCIETY FOR CRIPPLED CHILDREN AND ADULTS, INC., Chicago, represented by Dean W. Roberts, M.D., Executive Director, and Jayne Shover, Associate Director

The exhibit lists various environments and facilities in which speech rehabilitation is given by the National Society affiliates. Appropriate literature will accompany the exhibit, and the booth will be manned by a professional consultant from the National Society.

Exhibit S-2

Is Blindness on the Increase?

AMERICAN FOUNDATION FOR THE BLIND, INC., New York, represented by M. Robert Barnett and Kathern F. Gruber, New York; Richard E. Hoover, M.D., Baltimore

The exhibit will consist of a very large backdrop map of the United States showing the most current estimate of the incidence of blindness, state by state. This will be supported by related literature and professional consultant service at the booth. There will also be an exhibit of low-cost optical aids. An ophthalmologist will be in attendance.

Exhibit S-3

Stapes Mobilization: Techniques, Related Otosclerotic Pathology and Results

FRED R. GUILFORD, M.D.; WILLIAM K. WRIGHT, M.D.; W. LEONARD DRAPER, M.D., and C. OLAF HAUG, Ph.D., Houston, Texas, representing the Medical Branch of the University of Texas, Galveston, Texas

The exhibit will demonstrate through translights the various steps in the technique of the stapes mobilization operation. Wax models (three times normal size) will be shown to demonstrate the various types of otosclerotic changes encountered in fixation of the stapes. A model demonstrating the normal stapes footplate will also be shown for comparison. Results of the stapes mobilization operation and refixation percentages, as related to the degree of otosclerotic involvement, will be reported.

Exhibit S-4

Anatomical Studies Related to Cancer of the Larynx

JOEL JAY PRESSMAN, M.D.; CHARLES MONELL, M.D., and MILDRED BURTZ SIMON, School of Medicine, University of California at Los Angeles

This exhibit concerns the relationship existing between the site and spread of cancer of the larynx and the anatomical arrangement of the submucosal compartments, lymphatic flow, and vascular channels of the organ. Experiments have been carried out with injection of dyes and radioisotopes in living dogs and pigs, cadaver larynges, and human subjects. The findings are correlated with clinical cases of cancer. Conclusions reached stipulate that the detailed anatomical arrangement of the structures of the larynx have a direct bearing upon decisions concerning the type of operation to be performed in cancer of the larynx.

Exhibit S-5

Television in Otorhinolaryngology

HANS VON LEDEN, M.D., and PAUL MOORE, Ph.D., Northwestern University Medical School and the William and Harriet Gould Foundation, Chicago

This exhibit demonstrates the use of television in otorhinolaryngology. The difficulties inherent in the examination of the ears, nose and throat by reflected light with a television camera have been resolved by the design of a special adapter. Frequent demonstrations of this equipment will enable the audience to view a typical examination on the television screen. The potential of television in the teaching of otorhinolaryngology and in research in this field is stressed.

Exhibit S-6

Ballistic Studies of Protective Lenses

H. W. ROSE, M.D., School of Aviation Medicine USAF, Randolph AFB, Texas, and GEORGE M. STEWART, Biophysics Division, Chemical Corps Medical Laboratories, Army Chemical Center, Maryland

Plain glass lenses, heat treated lenses, laminated lenses and plastic lenses were subjected to impact by small high speed missiles. The results of these experiments necessitate a re-evaluation of industrial and military eye protection. The exhibit shows photographs, actual lenses, lens breakage patterns, diagrams and text.

Exhibit S-7

Tympanoplasty in 3-D

J. BROWN FARRIOR, M.D., Tampa, Florida

The surgical pathology of the middle ear is demonstrated in a series of 3-D photographs. The variations illustrate the indications and techniques for tympanoplasties, types I, II, III, IV, and V. Detailed interpretation of the photopathology is described in each case, demonstrated in a series of 200 slides in hand-operated individual stereoviewers. The basic types of tympanoplasties are illustrated in enlarged transparencies. A photographic record of the surgical pathology is advocated to demonstrate the reasons for success and failure, and thereby to develop sound judgment.

Exhibit S-8

Experimental Surgery of Stapes and Oval Window

RICHARD J. BELLUCCI, M.D., and DOROTHY WOLFF, Ph.D., Manhattan Eye, Ear and Throat Hospital, New York

Using experimental animals (monkeys and cats), various injuries were inflicted on the incudostapedial joint, crura of stapes and stapediovestibular joint. In some cases the stapes was completely removed, and in others it was disarticulated and replaced. The animals were then sacrificed after varying intervals of time. Local repair and consequences in the inner ear were studied.

Exhibit S-9

The Fundus Picture in External Exudative Retinopathy

H. SAUL SUGAR, M.D., Sinai Hospital and Wayne University College of Medicine, Detroit

Approximately twenty photographs of fundus changes in cases of Coats's disease and miliary aneurysms will be shown to indicate the similarities in these conditions. They will be exhibited in special viewers to give a pseudodepth appearance to the picture.

Exhibit S-10

Siderosis and Hemosiderosis Oculi (An Experimental and Clinical Study)

PAUL A. CIBIS, M.D.; TSUYOSHI YAMASHITA, M.D., and WILLIAM MOOR, Department of Ophthalmology, Washington University, St. Louis

A series of colored photomicrographs demonstrates the diffusion roots and affinity of ocular tissue components to iron ions of siderogenous or hemosiderogenous origin. The diffusion roots and binding of iron to mucopolysacharrides and other components in the eye after intravenous, anterior chamber, or vitreous body injections are illustrated at various phases. The early and late changes of siderotic or hemosiderotic origin and their implications to clinical observations of related origin will be emphasized.

Exhibits S-11

New Eyes for the Needy, Inc.

NEW EYES FOR THE NEEDY, INC., Short Hills, New Jersey, represented by Mrs. James S. Van Derbeck, Chatham, New Jersey; and Mrs. John H. Laporte and Mrs. Fred Emerson of Short Hills

New Eyes for the Needy, Inc., is a nonprofit volunteer charity dedicated to providing better vision to needy people both in this country and abroad. No money is solicited; the work is made possible through reclamation of discarded eye glasses and precious-metal scrap. New prescription glasses and artificial eyes are given to individuals screened by welfare agencies. Re-usable plastic framed glasses are tested and sent to hospital clinics and foreign missions. Funds have been established with 67 hospitals, and many welfare agencies are allotted specific numbers of glasses per year.

Exhibit S-12

Detection of Eye Problems Among Preschool Children

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS, New York, represented by Franklin M. Foote, M.D., Executive Director, Virginia Smith Boyce, Assistant Director, and Ruby M. Hopkins

The organization, operation and management of a community-wide preschool vision screening program will be described through photographs, posters and other illustrations. The procedure for training volunteers to do the screening and arrangements for follow-up care for children in whom there is an indication of the need for a professional examination will be outlined. Statistical

data from projects in which more than 30,000 preschool children have been tested will show the prevalence of eye trouble in this age group.

Exhibit S-13

Surgical Exposure of the Facial Nerve

STEPHEN P. HOGG, M.D., Cincinnati, Ohio, and ROBERT C. KRATZ, M.D., New Port, Kentucky, representing the University of Cincinnati College of Medicine, Cincinnati

A complete knowledge of the anatomic relationships of the facial nerve is necessary for safe and effective surgical operations on the temporal bone and parotid gland, and other surgical procedures in the upper neck and posterior mandibular area, as well as surgical exposure of the nerve itself.

After the surgeon has positively identified the facial nerve, he proceeds with safety and greater effectiveness in these anatomic areas. This exhibit presents a surgical approach which consistently identifies the facial nerve. This method is precise and constant because it refers to constant anatomic landmarks.

The exhibit consists of 8 × 10 colored photographs of surgical anatomy with appropriate legends and descriptions.

Exhibit S-14

The Spread of Infection in the Head and Neck

ORION H. STUTEVILLE, D.D.S., M.D., Northwestern University Medical School, Chicago

The exhibit consists of a series of photographs of patients with various space infections, as well as photographs of the anatomy involved. It demonstrates the spread of infection from its portal of entry into the various spaces of the head and neck.

Exhibit S-15

New Equipment for Military Ophthalmology

CAPTAIN BUDD APPLETON, MC, USA, Walter Reed Army Hospital, Washington, D. C.

A demonstration of field equipment, to include the ophthalmic field chest and a visual screening device.

Exhibit S-16

The Histopathology of the Ear in Conductive Deafness

JOSEPH G. DRUSS, M.D.; SIDNEY S. FEUERSTEIN, M.D., and JOSEPH L. GOLDMAN, M.D., The Mount Sinai Hospital, New York

A revived interest in the problems of conductive hearing impairment has developed in recent years because of the new surgical procedures such as fenestration, stapedial surgery, tympanoplasty and operations for congenital malformations which are now available for the treatment of certain types of conductive deafness. This exhibit will show histopathologic colored photomicrographs of the common conditions which interfere with the transmission of sound from the external and middle ears to the inner ear. The various causes of conductive deafness and pertinent clinical features will be presented on charts. Special reference will be given to otosclerosis.

Exhibit S-17

Processing the Whole Eye in Paraffin

ARMED FORCES INSTITUTE OF PATHOLOGY, Washington, D. C., represented by Lawrence P. Ambrogi

The exhibit will depict by photographs and specimens the various steps in preparing the eye for microscopic examination using the paraffin technique. This technique will be demonstrated by actual sectioning and staining by the exhibitor.

Exhibit S-18

Selection of Suitable Cases for Orthoptic Training

AMERICAN ASSOCIATION OF ORTHOPTIC TECHNICIANS, represented by Margaret Crush, Cincinnati; Glendon G. Smith, Hammond, Indiana; Nina Pick, Indianapolis, and Elizabeth May, Racine, Wisconsin

The aims of orthoptic training will be demonstrated by means of the establishment of single binocular vision. These aims can only be achieved through careful selection of cases.

Exhibit S-19

Medical Otolaryngology and Ophthalmology

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY and the HANSEL FOUNDATION, St. Louis, represented by French K. Hansel, M.D.

With the diminishing indications for the ordinary surgical procedures, there is evidence of an increasing interest in medical otolaryngology and ophthalmology. The exhibitors are interested, therefore, in the promotion of greater attention to medical subjects such as allergy, immunology, endocrinology, nutrition, headaches and other vascular phenomena in the graduate curriculum as well as in the private practice of the specialties.

Exhibit S-20

Glaucoma Research Project

THE OPHTHALMOLOGICAL FOUNDATION, INC., New York, represented by Conrad Berens, M.D., Managing Director, and Charles P. Tolman, Kew Gardens, New York

The purpose of this exhibit is to direct attention to the importance of early diagnosis of chronic simple glaucoma. Three avenues of the over-all problem are stressed: the high incidence of cases, the methods of diagnosis, and the position of the general man in the diagnostic picture wherein the probable glaucomas may best be detected. An instructional film in tonometry and ophthalmoscopy has been developed for medical schools and is generally available to medical societies or to individual physicians. An instrument designed to demonstrate the fallacy of finger palpation for low rises of pressure, as in early stages of chronic simple glaucoma, will be exhibited.

Exhibit S-21

Technique for Removing Temporal Bones for Pathological Study and Anatomical-Surgical Dissection

SUBCOMMITTEE ON RESEARCH OF THE COMMITTEE ON CONSERVATION OF HEARING, AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The exhibit shows the technique for removing temporal bones at autopsy, ways of minimizing postmortem degeneration and the method of preserving and mounting the temporal bone for surgical and anatomic dissection.

Exhibit S-22

Extended Septal Surgery

MAURICE H. COTTLE, M.D., Chicago Medical School, Chicago

Prepared anatomic specimens, illustrations and photographs demonstrate:

1. The maxilla-premaxilla approach in difficult anterior septum deformity corrections
2. Surgical methods of correction of complications and sequelae of septum operations
3. The septum approach to surgical correction of some pyramid and lobule deformities.

Exhibit S-23

Textbooks for the Visually Handicapped

AID TO VISUALLY HANDICAPPED, San Francisco, represented by Maurice D. Hart, M.D., Mrs. Gene Marchi, and Mrs. Maurice D. Hart

The exhibit is devoted to demonstration of large-type printed textbooks which have been transcribed into 18 point type from normal print books used in the public schools in California. The work is done by volunteers of AID TO VISUALLY HANDICAPPED, and distributed, free of charge, to all school districts and public libraries for the express purpose of aiding partially sighted children who are unable to read the smaller print textbooks.

Diseases of the Macula: Basic and Pathologic Changes Modifying the Clinical Picture

BERTHA A. KLIEN, M.D., Section of Ophthalmology, The University of Chicago

TECHNICAL EXHIBITORS — 1958

Exhibitor	Booth No.
Alcon Laboratories, Inc.	58 and 59
The Alkalol Company	29
Ambco, Inc.	R-35
American Cyanamid Company	R-16
American Optical Company	7 through 14
American Tobacco Company	R-17 and R-18
Armco Mercantile Company	R-23
Armorlite Lens Company	R-47
Artificial Human Eyes	R-21
Aurex Corporation	R-20
Austin Belgard, Inc.	18 and half of 19
Ayerst Laboratories	R-25
Barnes-Hind Laboratories, Inc.	R-49
Barnett Optical Laboratories	37
Bausch & Lomb Optical Company	95 through 102
Beltone Hearing Aid Company	R-9
Benson Optical Company	85, 86, 87
Blakiston Division, McGraw-Hill Book Co.	62
Boll & Lewis Optical Company	43 and 44
Broemmel Pharmaceuticals	63
Burroughs Wellcome & Co. (U.S.A.) Inc.	120
Cameron Surgical Specialty Company	76
Carroll Dunham Smith Pharmacal Co.	R-22
Chicago Reference Book Co.	R-2
ClerSite Company	A
The Coca-Cola Company	26
Codman & Shurtleff, Inc.	R-41 and R-42
Contact Lens Society of America	R-32
Continental Optical Company	R-15
Coreco Research Corp.	61
Curry & Paxton, Inc.	R-44, R-45, R-46
Joseph K. Dennis Company, Inc.	51
The DeVilbiss Company	16
Doho Chemical Corporation	34
Edwards Optical Company	R-30
Encyclopaedia Britannica	47
Ethicon, Inc.	64
Grason-Stadler Company	R-36
Grieshaber Swiss Eye Instruments	38
Grune & Stratton, Inc.	35
Guardian Chemical Corporation	R-19
Guild of Prescription Opticians of America, Inc.	41 and 42
Harlequin Corporation	6
Paul B. Hoeber, Inc.	32
Hollister-Stier Laboratories	46
The House of Vision, Inc.	65 through 68
Hudson Optical Corporation	109
Industrial Acoustics	R-37
Ion Optical Company	R-11 and R-12
The Iso-Sol Company, Inc.	R-43
Fritz Jardon Laboratories	R-6
Jenkel-Davidson Optical Company	105 and 107
Keeler Optical Products, Inc.	92 and 93A
Kelley & Hueber Co.	R-8
Keystone View Company	23 and 24
Kono Manufacturing Company	108
Lea & Febiger	53
Lederle Laboratories Division, American Cyanamid Company	R-10
Liberty Optical Mfg. Co., Inc.	90A
J. P. Lippincott Company	49

Exhibitor	Booth No.
Mager & Gougelman, Inc.	20 and half of 19
Maico Electronics, Inc.	69 and 70
Matalene Surgical Instruments Co., Inc.	81 and 82
McLeod Optical Company, Inc.	R-48
Medical Case History Bureau	115
Medical-Dental Photo Company	121
Medical Film Guild, Ltd.	L-L
Merck Sharp & Dohme, Division of Merck & Co., Inc.	45
Miller Surgical Company	89
Modern Optics, Inc.	118 and 119
The C. V. Mosby Company	88
V. Mueller & Company	1 through 5
Mueller Welt Contact Lens Laboratory	R-39 and R-40
New Era Optical Company	R-3 and R-4
Obrig Laboratories, Inc.	110
Ophthalmos, Inc.	R-13
The Opticase Company	15
Parsons Optical Laboratories	R-24
George P. Pilling & Son Co.	77, 78, 79
Plastic Contact Lens Company	R-1
Alfred P. Poll	R-27 and R-28
Precision-Cosmet Company, Inc.	116
W. F. Prior Company, Inc.	106
Professional Pharmacal Company	R-33
The Professional Press, Inc.	52
Radium Chemical Company	30
R. J. Reynolds Tobacco Company	27 and 28
Ritter Company, Inc.	71, 72, 73
Sandoz Pharmaceuticals	R-26
Schering Corporation	33
Schieffelin & Company	50
Robert B. Scott	R-34
Shuron Optical Company, Inc.	83 and 84
Smith, Kline & French Laboratories	R-29
Sonotone Corporation	80
E. R. Squibb & Sons	R-38
Storck Pharmaceuticals	25
Storz Instrument Company	111 through 114
Surgical Mechanical Research, Inc.	90B and 91
The Thermosector Company	31
Titmus Optical Co., Inc.	57
Uhlemann Optical Company	54, 55, 56
The Univis Lens Company	39 and 40
Victory Optical Mfg. Co.	103 and 104
The Vision-Ease Corporation	60
Dr. Max M. von Stirne	R-7
Walton Laboratories, Inc.	R-14
Warner-Chilcott Laboratories	R-5
Welch Allyn, Inc.	17
White Laboratories, Inc.	R-31
The White-Haines Optical Co.	21 and 22
The Williams & Wilkins Company	36
Winthrop Laboratories	117
Carl Zeiss, Inc.	74 and 75
Zenith Radio Corporation	93B and 94
Zylite Products Co., Inc.	48

TECHNICAL EXHIBITS

The Academy is appreciative of the cooperation of all technical exhibitors.

Booths with numbers preceded by "R" are located in the Red Lacquer Room.

Alcon Laboratories, Inc., Forth Worth
Booths 58 and 59

New developments in ophthalmic and rhinologic medication resulting from continuous research exclusively in these fields may be discussed with personnel from Alcon's Research and Sales Departments. Alcon's full line of sterile ophthalmic preparations for prescriptions, office treatment and ocular surgery will be displayed, together with Alcon-efrin, a specially formulated nonstinging, nonirritating nasal decongestant in three strengths.

The Alkalol Company, Taunton, Mass.
Booth 29

The Alkalol Company is featuring Alkalol, a solution especially designed for the treatment of the mucous membranes of the nose and throat.

First compounded in 1896, its balanced formula of salts and essential oils makes a solution that is effective and at the same time nontoxic, mild and pleasant. It is alkaline and a mucus solvent.

Alkalol is an excellent vehicle for many drugs. For further information please call at Booth 29.

Ambco, Inc., Los Angeles **Booth R-35**

You are cordially invited to inspect our line of audiometers, speech training equipment, and hearing aids. Competent personnel will be on hand to answer your questions and offer help.

American Cyanamid Company, Surgical Products Division, Danbury, Conn.
Booth R-16

We plan to display the Elliptron needle, a major advance in surgical needle design, Davis & Geck brand sutures featuring the emergency suture kit, with Surgilope Sterile Strip-Pack. Also VIM interchangeable hypodermic syringes and needles, including Gabriel vial aspirating syringe, VIM clear barrel interchangeable syringe, and a new, sterile-packaged Damascus hypodermic needle will be shown. Also displayed will be the Carver ring dispenser, Aureomycin dressings, Melacast orthopedic bandages, and a complete line of all surgical sutures.

American Optical Company, Southbridge, Mass. **Booths 7 through 14**

American Tobacco Company, Research Laboratory, Richmond, Va.
Booths R-17 and R-18

Armco Mercantile Co., Chicago
Booth R-23

Again exhibiting our exclusive specialties: operating and examination loupes of spectacle type, leaving both hands free, affording 2× and 3× telescopically clear magnification from eight inches to arm's length. For the first time we will exhibit 3× and 4× telescopic loupes with lenses encased in plastic tubes, these being the newest design of Henri Renault of Domont, France. Also on exhibit will be French and German first quality binoculars direct from importer to you, Graefe trial frame from Germany, and Mor-Vue fitting table fixture.

Armorlite Lens Company, Pasadena, Calif. **Booth R-47**

The Armorlite Lens Company, developers of durable hard resin lenses, will demonstrate the wide range in which light-weight, safe lenses are now available.

These will include examples of the safest of all lenses for children, athletes and industrial workers; of feather-weight aphakic bifocals; and of ultraviolet-ab-

sorbing hard resin lenses. Ultraviolet-absorbing lenses are crystal white and more perfectly transparent than glass to all light useful in seeing, yet they totally absorb all deleterious ultraviolet rays.

Artificial Human Eyes Booth R-21

Joint exhibitors in this booth are E. & S. Danz, New York; Greiner & Mueller, Chicago; Erickson Laboratories, Seattle; Erickson Laboratories, Spokane; Hugo Leipold, Pittsburgh; G. Danz & Sons, San Francisco; Earle C. Schreiber, Inc., Newark, N. J., and L. & B. Laboratories, Inc., Fort Lauderdale, Fla.

This exhibit of Artificial Human Eyes will include color photos of unusual cases. An implant and conformer for every type and preference will also be found here, and we shall repeat an exhibit of causes of frequent "unsolvable" irritations from ocular prostheses. We will also be on hand to discuss individual fitting problems with sincerity and humility.

Aurex Corporation, Chicago Booth R-25

Ayerst Laboratories, New York
Booth 25

The Ayerst exhibit will feature PREMARIN Intravenous, a physiologic hemostat, which has been used extensively to control hemorrhage promptly and safely following adenoidectomy and tonsillectomy. Also featured is THERUHISTIN, regular or S.A., which challenges comparison on all counts wherever antihistamine therapy is indicated. Ayerst representatives will be pleased to discuss these or other Ayerst products and make available the most recent clinical data.

Barnes-Hind Laboratories, Inc., Sunnyvale, Calif. Booth R-49

Barnett Optical Laboratories, Chicago
Booth 37

Bausch & Lomb Optical Co., Rochester, N. Y. Booths 95 through 102

A new super-flash Retinal Camera taking large colored pictures will be introduced. The new flash lamp is adaptable to all models of Bauch & Lomb Retinal Cameras.

The new Superscopic Greens' Refractor in all of the attractive colored finishes will be introduced. All dials and axis scales are much easier to read.

These new instruments will be the "features" of the complete display of Bausch & Lomb ophthalmic instruments.

The Cataract Lenticular Bifocal and the Panoptik Functional Trifocal will be displayed. Also shown will be the latest-in-fashion frames on Harmony Frame Bars. The display will include fitting room furniture.

Austin Belgard, Inc., Chicago
Booths 18 and 19

Beltone Hearing Aid Company, Chicago
Booth R-9

Benson Optical Company, Minneapolis
Booths 85, 86, 87

The Blakiston Division, McGraw-Hill Book Co., Inc., New York Booth 62

The Blakiston Division of McGraw-Hill Book Company will feature Paton, Katzin, and Stilwell's ATLAS OF EYE SURGERY. This text is brief, and all information, where possible, is illustrated by 464 extraordinarily fine and detailed line drawings of surgical procedures, all drawn from the point of view of the operating surgeon and clearly indicating intended procedures.

Also on display will be the new third edition of Harrison's PRINCIPLES OF INTERNAL MEDICINE; Arruga's OCULAR SURGERY, second English edition, featuring the new techniques and the most interesting modifications of the old ones which have appeared in recent years; Paton's KERATOPLASTY covering case selection, techniques, healing, and eye bank; Perry's OUR WONDERFUL EYES, a book for children.

Boll & Lewis Company, Chicago
Booths 43 and 44

Broemmel Pharmaceuticals, San Francisco Booth 63

Burroughs Wellcome & Co. (USA), Inc., Tuckahoe, N. Y. Booth 120

NEW PRODUCTS

The extensive research facilities of "B. W. & Co.," both here and in other countries, are directed to the development of improved therapeutic agents and techniques. Much basic theoretical work in our laboratories and in cooperation with internationally known institutions is contributing to the reservoir of fundamental medical knowledge.

Through such research "B. W. & Co." has made notable advances related to leukemia, malaria, diabetes, and diseases of the autonomic nervous system; and to antibiotic, muscle-relaxant, antihistaminic, and antinauseant drugs.

An informed staff at our booth will welcome the opportunity to discuss our products and latest developments with you.

Cameron Surgical Specialty Co., Chicago Booth 76

Fiat Lux (Let There Be Light) and there was light—and it was created by miniature lamps attached to various instruments made by the Cameron Surgical Instruments Company. More light than has ever before been seen is now in a brand-new safety (operating room) headlight being shown. The amazing thing is that the light is cold, using only 6 volts of electricity. In addition, we have about one hundred other instruments which closely approximate daylight illumination. The small (17 pounds) Model 550 portable electrosurgical unit for eye, ear, nose and throat work is the world's finest, yet the least expensive. It has been completely redesigned and is now available in a choice of colors. Two new operating prism loupes are being shown for the first time.

Carroll Dunham Smith Pharmacal Co., New Brunswick, N. J. Booth R-22

Our representatives desire to discuss continued clinical studies during the last twelve months that have added facts of interest to the ophthalmologist relative to the use of LIPOTRIAD capsules and liquid in connection with LIPOTRIAD therapy of certain retinopathies. LIPOTRIAD provides generous amounts of lipotropic agents and the oxytropic principles of B-complex vitamins.

Chicago Reference Book Co., Chicago Booth R-2

ClerSite Company, Chicago Booth A

The Coca-Cola Company, Atlanta Booth 26

Ice-cold Coca-Cola served through the courtesy and cooperation of the Coca-Cola Bottling Co., of Chicago, Inc., and the Coca-Cola Company.

Codman & Shurtleff, Inc., Boston Booths R-41 and R-42

Codman & Shurtleff, Inc., makers of fine surgeon's instruments since 1838, will exhibit their line of fine eye instruments, including the Beaver eye knives with detachable blades and a complete line of instruments for bronchoesophagology, together with accessory items.

The ear, nose and throat field will be represented by many standard and new items.

Contact Lens Society of America, Philadelphia Booth R-32

The Contact Lens Society of America, in its fourth year, will display a map showing the locations of its members. A booklet on the HISTORY OF CONTACT LENSES and other literature will be available to the members of the Academy. Members of the Society manning the booth will be ready to give information and answer questions about the Society and its members and discuss new developments in the field of contact lens fitting.

Continental Optical Company, Indianapolis Booth R-15

Coreco Research Corporation, New York Booth 61

The Coret Camera embodies the principle of electronic flash and constant automatic control of such factors as distance, aperture, field, and exposure. Now, for the first time, Coreco offers a completely automatic professional clinical

camera purposely designed to achieve the ultimate in surface, intraoral, and intratubular photography. Because of the simplicity of operation, even an inexperienced doctor or nurse can consistently achieve perfect color transparencies.

Curry & Paxton, Inc., Albertson, L. I., N. Y. Booths R-44, R-45, R-46

Curry & Paxton will feature the new Curpax Synoptiscope no. 10, designed to permit training at normal, everyday viewing conditions. The Dudragne Retinal Camera with stereoscopic sighting and built-in compensation for degree of ametropia of the patient and 1/800 second strobe flash. A field of 28° to 32° is covered and photographed at a magnification of X30 on standard 35 mm. film, giving an image 24 mm. square.

The Curpax Vanderbilt Projection Perimeter with direct recording; trial sets, trial frames and other ophthalmic aids, as well as the complete Curpax line of orthoptic equipment, will be on display.

Joseph K. Dennis Company, Inc., Chicago AAOO Group Insurance Administrators Booth 51

The AAOO Group Insurance Administrators (Joseph K. Dennis Company, Inc.) will be available in booth 51 to counsel with members concerning their membership privileges.

Plan to receive your "membership privilege packet." (Accident-Sickness Income Protection—Accidental Death, Dismemberment and Loss of Eyesight Protection—Professional Liability Protection—Professional Overhead Expense Protection—Major Hospital—Group Life and Retirement Annuity)

The DeVilbiss Company, Somerset, Pa. Booth 16

Doho Chemical Corporation, New York Booth 34

Doho Chemical Corporation is pleased to exhibit AURALGAN, the ear medication for the relief of pain in otitis media and removal of cerumen; OTOSMOSAN, the effective, nontoxic ear medication which is fungicidal and bactericidal (Gram negative-Gram positive) in suppuration and aural dermatomycoses; RHINALGAN, the nasal decongestant which is free from systemic or circulatory effect and equally safe to use on infants and on elderly people; and the NEW LARYLGAN, the soothing throat spray and gargle for infectious and noninfectious sore throat involvements.

Edwards Optical Company, Chicago Booth R-30

Encyclopaedia Britannica, Chicago Booth 47

A New Edition of the world-famous Encyclopaedia Britannica and its co-related services will be on display for the inspection of members and exhibitors at this meeting. The Encyclopaedia will be available at this meeting on a special discount basis.

Ethicon, Inc., Somerville, N. J. Booth 64

Grason-Stadler Company, West Concord, Mass. Booth R-36

Grieshaber Swiss Eye Instruments, Chicago Booth 38

For those who appreciate the finest in Ophthalmic Instruments we will display exclusive GRIESHABER patterns of EYE KNIVES, CORNEAL SUTURE NEEDLES, TREPHINES, and extremely delicate FORCEPS and NEEDLE HOLDERS.

Well known for their superlative cutting qualities, GRIESHABER EYE INSTRUMENTS and CORNEAL NEEDLES are made of a very special stainless steel which holds the original keen cutting edge.

With proper care, GRIESHABER INSTRUMENTS always prove to be a most economical investment.

We urge those who are not familiar with our instruments to purchase a trial order; the use of these instruments will enhance your operative technique, and their extreme sharpness and superior stainless qualities promote faster healing.

We take this opportunity to thank our many friends of the American Academy of Ophthalmology and Otolaryngology for their loyal support throughout the years and extend our invitation to all members to visit our display.

Grune & Stratton, Inc., New York
Booth 35

Guardian Chemical Corporation, Long Island City, N. Y. Booth R-19

The company will exhibit its CLOR-PACTIN WCS-90 for use in antibiotic-resistant eye, ear, nose and throat infections.

Also, there will be exhibited CLOR-PACTIN XCB as an effective adjunct to cancer surgery in the destruction of viable tumor cells dislodged during the operation.

Guild of Prescription Opticians of America, Inc., New York
Booths 41 and 42

Adjustment of glasses and minor repairs will be continuously available for the convenience of Academy members and guests who may need this service. Guild materials in support of medical eye care will be on display.

Harlequin Corporation, New York
Booth 6

Harlequin Corporation will present latest fashion eyewear featuring new seasonal colors and designs created to tie in with latest fashion trends.

Paul B. Hoeber, Inc., New York
Booth 32

Among the most important books of the year are the first three volumes of the second edition of Pack and Ariel's THE TREATMENT OF CANCER AND ALLIED DISEASES. Volume 3, TREATMENT OF TUMORS OF THE HEAD AND NECK, is of particular interest to members of the Academy. Other new works include the second edition of Sugar's THE GLAUCOMAS and Martin's SURGERY OF HEAD AND NECK TUMORS. If you do not already own the two classic volumes of Berliner's BIOMICROSCOPY OF THE EYE and Hollinshead's ANATOMY FOR SURGEONS, Volume 1: The Head and Neck, we hope you will accept our invitation to inspect them, as well as Sorsby's MODERN TRENDS IN OPHTHALMOLOGY, Third Series. All Hoeber-Harper books will be on display, and you are cordially invited to browse at leisure.

Hollister-Stier Laboratories, Philadelphia
Booth 46

A set of selected antigens for the Ophthalmologist and Otolaryngologist will be displayed.

New Reaction Guides for the scientific interpretation of diagnostic allergy reactions will be available to the interested physician.

Technical personnel will be in attendance at all times to consult with the physician on the HOLLISTER-STIER simplified techniques in allergy.

The House of Vision, Inc., Chicago
Booths 65 through 68

We will display the new diathermy unit developed by Dr. Charles Schepens and his associates at the Retina Foundation.

The production model of the Goldmann Aplanation Tonometer (Haag-Streit) is now available and can be mounted on any Haag-Streit slit lamp.

Again we will display the complete line of Haag-Streit precision instruments: Goldmann Slit Lamp, Goldmann Perimeter, Goldmann-Weekers Dark Adaptometer, Pfluger Perimeter, etc.

Zeiss equipment: Zeiss-Oberkochen Slit Lamp, Zeiss Refractionometer, Zeiss Fundus Camera.

Our display will include the very finest of European and American instruments, as well as the latest in technical optics and frame styling.

Hudson Optical Corporation, Long Island City, N. Y. Booth 109

Industrial Acoustics Company, Inc., New York Booth R-37

Ion Optical Company, Detroit
Booths R-11 and R-12

The Iso-Sol Co., Inc., Lindenhurst, N. Y. Booth R-43

The Iso-Sol Co. will exhibit its new improved Dropperettes, which are now prepunctured, sterile on all surfaces, and ready for immediate use. They will also display their new sterile OIL solutions of atropine, eserine and Pilocarpine. These oil solutions are less sensitizing and ideally adapted for nocturnal medication. Leakproof Lacrivials with self-sterilizing dropper tips and sterile Dacriose irrigators will be demonstrated.

Sulfa-Statin, a new decongestant ophthalmologic sulfonamide, will be featured among other new products. David J. Mason, Robert J. Brown, and George J. Prochaska will be in attendance and look forward to visiting with you.

Fritz Jardon Laboratories, Detroit Booth R-6

Jenkel-Davidson Optical Co., San Francisco Booths 105 and 107

You are invited to inspect specialized equipment and accessories designed and manufactured by Jenkel-Davidson Optical Company. The exhibit will include the latest model of the Clinicamera, Multiple Pattern Screener, Trial Lens Cabinet, Selectachart, Electrajust and other convenient accessories for the ophthalmologist.

Keeler Optical Products, Inc., Philadelphia Booths 92 and 93-A

Further developments of the Keeler Magnifying Spectacle now give surgeons a wider choice of magnification (1-3/4× to 9×) and working distance (15 inches to 7 inches) than ever before.

A flame-proof operating lamp with intense 12 watt beam or slit image is available as hand or stand model.

For subnormal vision patients the new Keeler aids provide magnification binocularly up to 5× at a constant and comfortable reading distance of 7.5 inches. With them, patients with 20/400 vision are enabled to read newsprint.

The Keeler "New Era" Dynamic Retinoscope has been still further improved by incorporating a focusing movement and providing even more brilliant illumination than hitherto.

Kelley & Hueber, Inc., Philadelphia Booth R-8

We are planning to exhibit a complete line of spectacle cases including soft cases of all qualities from the finest saddle leathers and top grain cowhides down through the plastics and imitation leathers.

We are also planning to exhibit a complete line of hard metal reinforced spectacle cases and many novelty items. We also expect to introduce at this time a new series of Contact Lens Kits and Cases that have been specifically designed with the Contact Lens patient in mind.

Keystone View Co., Meadville, Pa. Booths 23 and 24

Kono Sales Co., Inc., Woodside, N. Y. Booth 108

Lea & Febiger, Philadelphia Booth 53

Lea & Febiger invites you to examine the many books of interest to members of the American Academy of Ophthalmology and Otolaryngology. Be sure to see Cushman—STRABISMUS; MacNeal, Alpers and O'Brien—HEADACHE; Ballenger and Ballenger—DISEASES OF THE NOSE, THROAT AND EAR; Town—OPHTHALMOLOGY; Krimsky—BINOCULAR IMBALANCE; Ballenger and Ballenger—A MANUAL OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY; Spaeth—OPHTHALMIC SURGERY; Cowan—REFRACTION OF THE EYE; Gifford—OCULAR THERAPEUTICS; Joslin—TREATMENT OF DIABETES MELLITUS; Wohl and Goodhart—NUTRITION; Bonica—MANAGEMENT OF PAIN; and many others.

Lederle Laboratories Division, American Cyanamid Company, Pearl River, N. Y. Booth R-10

Liberty Optical Manufacturing Co., Inc., Newark, N. J. Booth 90-A

Visitors to the Liberty Optical Manufacturing Company booth will see the season's most exciting new feminine

frame creation—the LIBERTE. Exotic in shape, bewitchingly beautiful in character, the new all-zyl LIBERTE will be prominently displayed in five striking colors: Black, Gunsmoke, Bluesmoke, Brownsmoke and White Pearl.

On hand to greet the Academy members and their guests will be Mr. Joseph G. Bianco, Liberty General Manager, Tony DiChiara, Secretary-Treasurer, and Jerry Weissman, sales representative.

J. B. Lippincott Company, Philadelphia
Booth 49

J. B. Lippincott Company presents, for your approval, a display of professional books and journals geared to the latest and most important trends in current medicine and surgery. These publications, written and edited by men active in clinical fields and teaching, are a continuation of more than 100 years of traditionally significant publishing.

Mager & Gougelman, Inc., Chicago
Booths 19 and 20

Complete Artificial Eye Service, Facial Restorations and Precorneal Lenses. Our skilled glass and plastic eyemakers are heirs to our 107 years of experience. We have offices in many principal cities, and we visit other localities regularly for the fitting of artificial eyes.

We provide extensive mail order service for doctors who must do their own fittings. FREE COLOR CHART AND SIZE GUIDE available at our booth, where you are also invited to see our implants, conformers, artificial eyes, anatomical eye models, facial restorations and precorneal lenses.

We will welcome the opportunity of discussing with you any artificial eye or lens problems you may have.

Maico Electronics, Inc., Minneapolis
Booths 69 and 70

Matalene Surgical Instruments Co., Inc., New York **Booths 81 and 82**

Matalene Surgical Instruments Co., Inc., will exhibit an improved binocular gonioscope with coaxial illumination, the Chamlin perimeter, tonometers, trial frames, and the newest developments in forceps scissors, cutting instruments, instrument cases, etc., as well as the standard patterns.

McLeod Optical Co., Inc., Providence, R. I. **Booth R-48**

The McLeod Optical Company will feature the new Aspheric Hyperocular Lenses for the subnormal visioned.

They will have a complete line of I-Gard Aspheric Readers and magnifiers. Also, the complete line of I-Gard Plastic ophthalmic lenses including the popular I-Gard Lenticular Cataract Bifocal.

Norman MacLeod, Ed McLeod and Norm MacLeod, Jr., will be in attendance at the booth.

Medical Case History Bureau, New York
Booth 115

Simplifying the Doctor's History Record and Bookkeeping System with the INFO-DEX RECORD CONTROL SYSTEM.

Maintenance of accurate, informative data on both history and financial records is essential in the modern doctor's practice. The INFO-DEX Record Control System helps to keep a constant finger on the physical and financial status of the patient. This system correlates information almost automatically for instant reference and research work. The method of cross-indexing interesting cases according to the disease is unique and exclusive.

The MEDICAL CASE HISTORY BUREAU of New York City has specialized for many years in record forms for the doctor's office. Its well-informed representative will gladly demonstrate the Info-Dex System and discuss your office problems.

Medical-Dental Photo Co., Levittown, N. Y. **Booth 121**

Featuring the highly successful Anterior Segment Camera with the Rotating Reflex Speedlight. This inexpensive set-up was unveiled for the first time in 1957 and the interest was tremendous. The low priced electronic Fundus Camera

from France, product of Optiques et Precision (O.P.L.), will also be shown. Stereo Eye Camera will be available, as will the slides taken with the stereoscopic close-up camera. For the Otolaryngologists, our Research Department is working hard on your problems, and we sincerely hope that we will have the answer for you in 1959.

Medical Film Guild, Ltd., New York
Booth L-L

Merck, Sharp & Dohme, Division of Merck & Company, Inc., Philadelphia
Booth 45

A new and very promising diuretic is featured at the Merck Sharp & Dohme booth. Since the principal action of DIURIL is a marked enhancement of the excretion of sodium, chloride and water, it has been designated a saluretic agent. This new compound achieves a profound electrolyte and water diuresis without attendant toxic effects and other disadvantages peculiar to the mercurials and certain other diuretic agents.

Technically trained personnel will be present to discuss this and other subjects of clinical interest.

Miller Surgical Company, Chicago
Booth 89

Modern Optics, Inc., Houston, Texas
Booths 118 and 119

The C. V. Mosby Company, St. Louis
Booth 88

V. Mueller & Company, Chicago
Booths 1 through 5

V. Mueller & Company will feature a broad selection of instruments for both stapes mobilization and fenestration procedures, in addition to the usual comprehensive display of fine instruments—both standard and special—for surgery of the eye, ear, nose and throat. The latest Shambaugh-Zeiss microscope will be demonstrated, as will other Mueller equipment.

Mueller Welt Contact Lenses, Inc., Chicago
Booths R-39 and R-40

We will introduce at this meeting the new TRI-CON lens, a tri-curve corneal micro lens of extreme comfort. This development, we feel, has come as close to the ultimate in a contact lens that is possible with present day plastics. Eighteen hundred patients have used this lens for one and a half years. Over 1,500 are all-day wearers. Less than 150 patients had contraindications for its use.

We will also show a unique development, our new bifocal contact lens—now being tried by 65 patients, and will report the results.

Be sure to attend one of the daily contact lens demonstrations at our booth.

New Era Optical Company, Chicago
Booths R-3 and R-4

Obrig Laboratories, Inc., New York
Booth 110

The Obrig Laboratories will display the latest types of fluidless lenses. Also on display will be a newly designed radius gauge for measuring the posterior curve of contact lenses, centering devices and thickness gauges. Also, there will be available, pamphlets, many new folders, textbooks, as well as slide rules, supplies and accessories.

Ophthalmos, Inc., Division of Doho Chemical Corp., New York Booth 34

You are cordially invited to visit our booth and become acquainted with the innovations now being offered by Ophthalmos, Inc.

Ophthalmos, Inc., is now a division of the Doho Chemical Corporation, with greatly augmented research facilities. As a result, many of the research projects of Ophthalmos have come to fruition.

We will exhibit the new STERI-TAINER, an Autoclaved STEAM STERILIZED container with controlled single drop plug in the new convenient flat oval shape.

At present we offer Almocarpine solutions, Vas I Zinc and Tetracaine in the STERI-TAINER. Other solutions will be offered when tests show that they are compatible with the container.

The new Fluor I Strip package containing 200 individually enveloped Fluor I Strips will also be shown.

A product of major importance to the ophthalmologist will be introduced . . . Ophthalmic Anhydrous Glycerol (Doho) made Hypertonic with 5% Sodium Chloride for the rapid reduction of Corneal Edema and the treatment of Bullous Keratitis.

Robert R. Feinstein, our Director of Research, will be available to discuss any questions you may have on the above products, plus Epitrate and Almocarpine.

The Opticase Company, Newark, N. J. Booth 15

Parsons Optical Laboratories, San Francisco Booth R-24

Parsons Optical Laboratories, the world's largest manufacturers of glaucoma equipment, will show both diagnostic and surgical instruments in that field, including the Barkan-Koeppe lens and the Barkan goniotomy lens. Also floor model gonioscopes, hand-held microscopes, the Barkan focal illuminator and operating light, and a complete display of tonometers.

In addition to glaucoma instrumentation, other items of our exclusive manufacture such as the Costenbader accommodometer and the Costenbader adjustable clip-on trial frame, Harrington pocket charts, Engel fundus contact lens, Thorpe fundus lens, and Thorpe x-ray localization lens. Also some equipment by Haag-Streit, whom Parsons represents as West Coast distributors.

George P. Pilling & Son Company, Philadelphia Booths 77, 78, 79

We will have on display many new and interesting items for the EENT specialist. Also on display will be a full display of the new Chevalier Jackson Clinic instruments for bronchoesophagology, as well as a new assortment of center action foreign body and biopsy forceps. See the new equipment for Foto bronchoscopy.

Plastic Contact Lens Company, Chicago Booth R-1

Mr. Alfred P. Poll, New York Booths R-27 and R-28

Precision-Cosmet Company, Inc., Minneapolis Booth 116

W. F. Prior Company, Inc., Hagerstown, Md. Booth 106

Professional Pharmacal Company, Inc., San Antonio, Texas Booth R-33

The Professional Pharmacal Company will display its complete line of BufOpto Ophthalmic Solutions.

Two NEW superb BufOpto ophthalmic solutions will be featured. BufOpto VERNACEL is a sterile decongestant and antihistaminic ophthalmic solution that provides prompt, effective relief in hay fever, vernal conjunctivitis, and many allergies. This solution contains no dangerous steroids.

BufOpto EFRICEL-10% is a stable ophthalmic solution containing Phenylephrine HCl. 10%. This unique preparation, the result of four years of research and testing, will not darken or discolor on long standing.

All BufOpto Ophthalmic Solutions will be featured in the New All-Plastic package that permits inspection of the contents of the package at all times.

Our special representatives will be on hand to answer any questions concerning these products.

The Professional Press, Inc., Chicago Booth 52

The Eye, Ear, Nose and Throat Monthly is now in its thirty-seventh year of publication. It is a practical journal whose aim is to present material of interest and value to the ophthalmologist and otolaryngologist. Sample copies will be available. THE RED BOOK—the directory of eye, ear, nose and throat specialists—will also be on display.

Radium Chemical Company, Inc., New York Booth 30

Radium Chemical Company, Inc., will display the Monel metal radium nasopharyngeal applicator, used for the treatment of hyperplastic lymphoid tissue at the orifice of the eustachian tube and the fossa of Rosenmueller. A complete line of instruments for the application of radon will also be shown.

R. J. Reynolds Tobacco Co., Winston-Salem, N. C. Booths 27 and 28

Welcome to the R. J. Reynolds Tobacco Company Exhibit! You are cordially invited to receive a cigarette case (monogrammed with your initials) containing your choice of CAMEL, WINSTON Filter, Menthol Fresh SALEM, or CAVALIER King Size Cigarettes.

Ritter Company, Inc., Rochester, N. Y. Booths 71, 72, 73

Sandoz Pharmaceuticals, Hanover, N. J. Booth R-26

Sandoz Pharmaceuticals cordially invites you to visit our display.

BELLERGAL Space Tabs assure around-the-clock control of functional complaints (example—menopause symptoms) in the periphery where they originate.

SANDOSTENE Space Tabs: around-the-clock control of itching and hay fever.

BepHAN Space Tabs: a new approach to prolonged maintenance of low gastric acidity.

Our representatives in attendance, will gladly answer questions about these and other Sandoz products.

Schering Corporation, Bloomfield, N. J. Booth 33

A cordial invitation is extended to the members of the American Academy of Ophthalmology and Otolaryngology to visit the Schering exhibit. The entire exhibit will be devoted to the use of the "Meti" steroids—METIMYD (corticoid-antibacterial combination), METICORTEN and METICORTELONE—in topical and systemic therapy of allergic and inflammatory disease of the eye. Extensive clinical and laboratory data demonstrating certain advantages of these steroids over cortisone and hydrocortisone are presented.

Schieffelin & Co., New York Booth 50

Schieffelin & Company will exhibit CYCLOGYL (Cyclopentolate hydrochloride). CYCLOGYL is indicated for Cycloplegia and Mydriasis, and provides maximum Cycloplegia and Mydriasis for the shortest duration.

The extensive advantages of CYCLOGYL, as well as the economy obtained through the use of CYCLOGYL for routine refractions, will be discussed by professional representatives.

Along with the CYCLOGYL exhibit, new ophthalmic and aural preparations will be discussed. Schieffelin & Company is presently planning to introduce several new preparations in these areas of medicine. Samples and literature on CYCLOGYL and other products will be available.

Robert B. Scott, Chicago Booth R-34

Shuron Optical Company, Inc., Rochester, N. Y. Booths 83 and 84

Smith, Kline & French Laboratories, Philadelphia Booth R-29

SKF features (1) TRISOCORT 'SPRAYPAK', containing hydrocortisone, 3 antibacterials and 2 decongestants for intranasal infections; (2) VASOCORT 'SPRAYPAK' AND SOLUTION, containing hydrocortisone and 2 decongestants for intranasal inflammation and congestion; (3) TELDRIN SPANSULE sustained release capsules, for rapid and sustained antihistaminic effect—all day or all night with a single oral dose; (4) COMPAZINE, the tranquilizer and antiemetic that is virtually free from drowsiness and depressing effect; and (5) THORAZINE, one of the fundamental drugs in medicine. Our representatives welcome the opportunity to discuss SKF products with you, and are ready always to be of help in any way they can.

Sonotone Corporation, Elmsford, N. Y.
Booth 80

This exhibit will feature the complete line of Sonotone audiometers, including models for the doctor's office, clinics, industry and schools. These audiometers meet or exceed the specifications of the American Standards Association in every respect and are designed to combine lasting accuracy with rugged dependability, yet all four models are offered at unparalleled low prices. Also shown will be the newest in Sonotone hearing aids, including "all-in-the-ear" and eyeglass models. The latter models include both air and bone conduction types.

E. R. Squibb & Sons, New York
Booth R-38

E. R. Squibb & Sons has long been a leader in the development of new therapeutic agents for prevention and treatment of disease. The results of our diligent research are available to the medical profession in new products or improvements in products already marketed.

At booth R-38, we are pleased to present up-to-date information on these advances for your consideration.

Storck Pharmaceuticals, St. Louis
Booth 25

ECTASULE: The first long-acting bronchodilator in timed disintegration capsule form. These capsules contain ephedrine and amobarbital to give complete nocturnal protection to patients with hay fever, vasomotor rhinitis and acute sinusitis. Available in two dosage forms.

PENTERGOT: "Our old standby" for treating migraine and tension headaches—in rectal inserts and oral tablets.

METALEX: Combines pentylenetetrazol and nicotinic acid to reactivate brain cells damaged by arteriosclerosis, and thereby improve visual acuity from 20/80 to 20/30 in a period of three weeks.

Storz Instrument Co., St. Louis
Booths 111 through 114

Featured in the Storz instrument display will be a complete selection of fine surgical instruments for the eye and ear, nose and throat, bronchoscopic and nasal plastic surgeons. All new instruments developed during 1958 will be featured.

Of particular interest to the otologist will be the sets of instruments for stapes mobilization and tympanoplasty, of both American and European designs. In addition, the exhibit will feature a comparative display of the ZEISS, AMERICAN OPTICAL, and STORZ SURGICAL MICROSCOPES.

Of importance to the ophthalmic surgeon will be many unique developments, including an electrically powered OPHTHALMIC DERMATOME for taking Lamellar Donor Grafts, as well as Tissue Grafts, for lid repairs.

Surgical Mechanical Research, Inc., Los Angeles **Booths 90B and 91**

An efficiently arranged office which is convenient in arrangement, economical in use of available space, and reasonable in cost, and which maintains its appearance of newness over the years will be on display.

Also shown will be the SMR EAR STOPPER, which is used in industry to prevent acoustic trauma. Literature will be furnished on request.

A new Eye Cabinet will be shown, complete with retractable electric cords for the ophthalmoscope and retinoscope, a warmer for eye solutions, facilities for illuminating the work area, and an additional bank of nine shallow drawers.

The Thermosector Company, Oak Park, Ill. **Booth 31**

The Thermosector, an electrosurgical unit expressly designed for eye, ear, nose and throat techniques in office and hospital, will be exhibited and demonstrated. You are invited to investigate the superior features of this device, which is preferred by leading hospitals, clinics and professional men.

See, also, the Dazor Floating Arm Magnifier Examining Lamps with no-glare fluorescent lighting on display at the Thermosector booth.

Titmus Optical Company, Inc., Petersburg, Va. **Booth 57**

Uhlemann Optical Company, Chicago
Booths 54, 55, 56

The Univis Lens Company, Dayton, Ohio
Booths 39 and 40

Victory Optical Manufacturing Co., Newark, N. J. Booths 103 and 104

The latest exhibit booths of Victory Optical Manufacturing Company will feature "the styles of tomorrow today," and the latest colors in fashion eyewear. All Victory frames are precision crafted and ophthalmically correct, exemplifying the most desirable features of fine fashion eyewear. The best quality and the fastest service is the password in every department.

The Vision Ease Corporation, St. Cloud, Minn. Booth 60

Vision-Ease will display its full line of improved type multifocals including the new Neutral Gray, the tints and greens. Special emphasis will be given to the Catarex service with the temporary and permanent lens forms. The constantly increasing use of this service for the fitting of postoperative cataract patients is evidence of the broad acceptance by both the patient and the doctor of a comfortable, complete, and yet economical solution to an otherwise complex and expensive procedure.

Dr. Max M. von Stirne, Philadelphia
Booth R-7

Walton Laboratories, Inc., Irvington, N. J. Booth R-14

Warner-Chilcott Laboratories, Morris Plains, N. J. Booth R-5

The White-Haines Optical Co., Columbus, Ohio Booths 21 and 22

The White-Haines Optical Company will exhibit the following instruments: Bausch and Lomb Deluxe Motor Refracting Unit, Compact Acuity Projector and the Greens' Refractor, all in the new Biscayne Blue color.

Other instruments and equipment displayed will be the new Reliance adjustable instrument table, Reliance hand operated hydraulic treatment chair, Bausch and Lomb Thorpe slit lamp, featuring the Bal-Cold reflector with a wide angle prism, Haag-Streit slit lamp, Airmark projector perimeter, Airmark trial frame, Bausch and Lomb keratometer and vertometer, along with a complete line of diagnostic instruments.

Welch Allyn, Inc., Skaneateles Falls, N. Y. Booth 17

White Laboratories, Inc., Kenilworth, N. J. Booth R-31

The Williams & Wilkins Co., Baltimore
Booth 36

Winthrop Laboratories, New York
Booth 117

Carl Zeiss, Inc., New York
Booths 74 and 75

Zenith Radio Corporation, Hearing Aid Division, Chicago
Booths 93B and 94

Zylite Products Co., Inc., New York
Booth 48

SECTION ON INSTRUCTION

SIXTY-THIRD ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

October 12-16, 1958

PALMER HOUSE

Chicago, Illinois

A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

DEAN M. LIERLE, M.D.
Secretary for Instruction — Otolaryngology and Maxillofacial Surgery

The following condensed schedules of instruction courses are reprinted for the convenience of Fellows who have not yet placed orders for tickets.

Courses already sold out are so designated on the charts. Please do not request tickets for these courses.

For complete listing of course titles, instructors, and descriptive paragraphs about each subject, please refer to the March-April issue of the TRANSACTIONS.

General Instructions

Only members who have paid their 1958 dues may order instruction tickets in advance of the meeting. Members should order tickets only for their own use. *Candidates for fellowship in the Academy are not privileged to reserve tickets in advance.* After the session opens, no distinction between members, candidates and guests will be made in selling tickets.

Each period (1 hour) of instruction costs $2.00. *Orders unaccompanied by properly executed checks will not be filled.* Checks should be made payable to W. L. Benedict, M.D.

In case of inability to attend the meeting after order has been filled, a refund will be granted if requested prior to October 10.

If courses requested are not available and it is impossible to substitute, a refund will be granted. Refunds will not be sent by mail. A blue refund card will be placed in the Advance Registration Envelope which is received at the time of registration. This should be presented at the Instruction Desk for payment.

All orders for tickets will be filled according to postmark on envelope containing check. Telegrams will not be honored. Orders will not be accepted for presale after October 2. To ensure acceptance they should be mailed by September 30 to William L. Benedict, M.D., 15 Second Street S.W., Rochester, Minnesota.

Ordering Instruction Tickets

An *individual* course is complete in one (1) period. Most individual courses are repeated on two different days, allowing the subscriber to choose the day on which he wishes to attend.

A *continuous* course runs two (2) or more periods as indicated. It is not possible to purchase a ticket to only *part* of a continuous course.

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful attention to instructions will ensure the correct filling of your requests. *Since number of tickets for each course is limited, be sure to list second and third choices for each period in case first choice is already sold out. In every case, effort will be made to furnish first choices.*

If you are requesting registration in the continuous courses, please specify preferences for individual courses as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Acknowledgment of Advance Order

As soon as an advance order is filled, a postcard, listing the courses that have been reserved for him, will be mailed to the subscriber. Please do not ask to change courses after your order has been filled. The postcard is merely an assurance that your order has been filled; it is not necessary for registration at the Academy meeting.

Claiming Instruction Tickets

No tickets will be sent through the mails. Members who have ordered instruction course tickets will register at the *Advance Order Section* of the Registration Desk during the week of the Convention to secure registration badge, tickets, and all convention material.

Tickets will be on sale during the meeting at the Instruction Desk in the Registration Foyer.

If you have ordered tickets in advance, at the time of registration take your registration card to the desk marked ADVANCED ORDERS. There you will be given an envelope containing badge, tickets and program which has been prepared ahead of time to facilitate your registration.

KEY TO DESIGNATION OF PERIODS

The periods will be designated with the following key: Select tickets for — Otolaryngology, forenoons, periods 1-2-3 — Ophthalmology, afternoons, periods 4-5-6

Day	(M)onday	(T)uesday	(W)ednesday	(Th)ursday
Period a.m.		1 2 3	1 2 3	1 2 3
Period p.m.	4 5 6	4 5 6	4 5 6	4 5 6

M-4	2:00 p.m. to 3:00 p.m.	W-4	2:00 p.m. to 3:00 p.m.
M-5	3:15 p.m. to 4:15 p.m.	W-5	3:15 p.m. to 4:15 p.m.
M-6	4:30 p.m. to 5:30 p.m.	W-6	4:30 p.m. to 5:30 p.m.
T-1	9:00 a.m. to 10:00 a.m.	Th-1	9:00 a.m. to 10:00 a.m.
T-2	10:15 a.m. to 11:15 a.m.	Th-2	10:15 a.m. to 11:15 a.m.
T-3	11:30 a.m. to 12:30 p.m.	Th-3	11:30 a.m. to 12:30 p.m.
T-4	2:00 p.m. to 3:00 p.m.	Th-4	2:00 p.m. to 3:00 p.m.
T-5	3:15 p.m. to 4:15 p.m.	Th-5	3:15 p.m. to 4:15 p.m.
T-6	4:30 p.m. to 5:30 p.m.	Th-6	4:30 p.m. to 5:30 p.m.
W-1	9:00 a.m. to 10:00 a.m.		
W-2	10:15 a.m. to 11:15 a.m.		
W-3	11:30 a.m. to 12:30 p.m.		

(Familiarity with this key will be helpful and will avoid confusion)

The Following are Condensed Schedules of Courses Previously Described

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
1	Allen et al.	Bacteriology and Infectious Diseases	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
2	Ascher	Aqueous Veins in Glaucoma	2 PERIODS M-4, M-5
3	Ballintine & Purnell	.....SOLD OUT.....	
4	Becker & Kinsey	Studies on Aqueous Flow	2 PERIODS T-4, T-5
5	Bedell	Ophthalmoscopy	3 PERIODS M-6, T-6, W-6
6	Boeder	Practical Optics	2 PERIODS M-5, M-6
7	Braley	.....SOLD OUT.....	
8	Breinin	Electromyography	2 PERIODS W-4, W-5
9	Brockhurst	Indirect Ophthalmoscopy	2 PERIODS T-5, T-6
10	Brown, H. et al.	Eye Muscles	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
11	Byrnes	Ophthalmic Filters	2 PERIODS T-5, T-6
12	Callahan et al.	Ophthalmic Plastic Surgery: Advanced	8 PERIODS M-5, M-6, T-5, T-6 W-5, W-6, Th-5, Th-6
13	Clark	Retinal Detachment	3 PERIODS M-4, T-4, W-4
14	Cooper	Principles of Muscle Surgery	2 PERIODS T-5, T-6
15 or 16	Copeland	Streak Retinoscopy	4 PERIODS M-4, M-5, T-4, T-5 or W-4, W-5, Th-4, Th-5
17	Costenbader	Muscle Problems	2 PERIODS M-4, M-5
18	Costenbader & Simpson	Probing Nasolacrimal Duct in Infant & Adult	2 PERIODS T-4, T-5
19	Dickey et al.	Ocular Motility	6 PERIODS M-5, M-6, T-5, T-6 W-5, W-6
20	Dunnington & Regan, E.	Cataract Surgery: Complications Due to Defective Wound Healing	2 PERIODS M-4, M-5
21	Fonda & Sloan	Subnormal Vision: Technique for Examination	2 PERIODS T-4, T-5
22	Gettes & Donnelly	Refraction	6 PERIODS M-4, M-5, T-4, T-5 W-4, W-5
23	Guibor	Orthoptics: Office & Home Techniques	3 PERIODS M-4, T-4, W-4
24	Harley	Lesions of 3rd, 4th & 6th Cranial Nerves	2 PERIODS W-5, W-6
25	Hughes et al.	Ophthalmic Plastic Surgery: Fundamental	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
26	Jones, L.	Lacrimal Drainage Apparatus	2 PERIODS M-4, M-5

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
27	Knighton et al.	Practical Essentials of Glaucoma	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
28	Lemoine et al.	Fundus Lesions	2 PERIODS M-5, M-6
29	Linksz	Physiology of Vision	2 PERIODS W-5, W-6
30	Masters, R., et al.	Slit Lamp Biomicroscopy: Fundamentals	9 PERIODS M-4, M-5, M-6, T-4, T-5 T-6, W-4, W-5, W-6
31	McLean & Guyton	.....SOLD OUT.....	
32	Mullen et al.	Ocular Emergencies	3 PERIODS M-4, T-4, W-4
33	Pischel et al.	Retinal Detachment	4 PERIODS M-5, M-6, T-5, T-6
34	Posner & Gorin	Office Gonioscopy	2 PERIODS T-4, T-5
35	Regan, C., & Boeder	Refraction	4 PERIODS M-4, M-5, T-4, T-5
36	Roberts	Glaucoma: Early Diagnosis	2 PERIODS M-4, M-5
37	Rolett	Gonioscopy: Practical Application	2 PERIODS W-4, W-5
38	Romaine	Ocular Motility Surgery	4 PERIODS M-4, M-5, T-4, T-5
39	Romaine	Refraction: Problems & Treatment	3 PERIODS M-6, T-6, W-6
40	Schepens et al.	Retinal Detachment	8 PERIODS M-5, M-6, T-5, T-6 W-5, W-6, Th-5, Th-6
41	Schimek & Bousquet	Clinical Gonioscopy & Tonography	2 PERIODS W-4, W-5
42	Schlaegel & Hungerford	Uveitis	2 PERIODS Th-4, Th-5
43	Shaffer	Gonioscopy: Practical Application	2 PERIODS T-5, T-6
44	Stow & Moss	Diagnostic & Therapeutic Office Procedures	2 PERIODS M-5, M-6
45	Theobald et al.	Histopathology	12 PERIODS M-4, M-5, M-6, T-4, T-5, T-6, W-4, W-5, W-6, Th-4, Th-5, Th-6
46	Thorpe	Intraocular Foreign Bodies	2 PERIODS M-4, M-5
47	Thorpe et al.	.....SOLD OUT.....	
48	Thorpe et al.	Slit Lamp Gonioscopy	6 PERIODS T-4, T-5, T-6 W-4, W-5, W-6
49	Townes	Cataract Surgery: Complications	2 PERIODS T-5, T-6
50	Troutman et al.	Recent Advances in Ophthalmology	8 PERIODS M-5, M-6, T-5, T-6 W-5, W-6, Th-5, Th-6
51	Troutman	Enucleation Surgery	2 PERIODS T-4, T-5
52	Wheeler & Knapp	Concomitant Strabismus	4 PERIODS M-4, M-5 T-4, T-5
53	Home Study		10 PERIODS M-4, M-5, M-6, T-4, T-5 T-6, W-4, W-5, W-6, Th-4

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Monday

Key M-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
105	Alvaro	Uveitis: Etiology & Treatment
110	Bannon	Aniseikonia
119	Calhoun	Epithelization of Anterior Chamber
122	Cassady	Toxoplasmic Retinochoroiditis
133	Cutler	Cataract: Methods of Extraction
136	Davis	Sutures in Cataract Surgery
138	DeVoe	Corneal Dystrophies & Degeneration
139	Diskan	Vision Screening in Schools
145	Everett	Retinal Detachment: Selection of an Operation
151	Fralick	Principles of Lid Surgery
152	Frayer	Retained Lens Protein After Operation & Trauma
181	Kornzweig	Clinical Embryology
200	Marks	Electrical Currents Used in Ophthalmology
205	Miller	Corneal Diseases
215	Payne	Extraction of Complicated Cataract

Key M-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
120	Carroll	Optic Nerve Lesions: Differential Diagnosis
137	Day	Medical Exophthalmos: Diagnosis & Treatment
142	Dunlap	Divergence Anomalies
148	Ferrer	Contact Lenses
154	Gartner	Fundus Diseases: Clinical & Pathologic Correlations
157	Gifford	Local Anesthesia with Special Reference to Deep Motor Block
161	Haik	Intracapsular Cataract Extraction
221	Reese	Cataract Extraction
226	Scarney	Slit Lamp (Biomicroscopy)

Key M-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
104	Allen, H.	Aseptic Technique in Ophthalmology
106	Alvis	Glaucoma Surgery: Long-Term End Results
111	Berens	Cycloelectrolysis, Cyclodiathermy, Iridencleisis & Cyclodialysis
114	Bettman	Radioactive Isotopes
127	Cholst	Central Fields: Methods & Techniques
128	Cohoe	Special Reading Instruction for the Partially Seeing
129	Coston	Inflammatory & Degenerative Disease of Retina
131	Cunningham	Types of Head Pain
134	Daily	Biomicroscopy of Vitreous & Fundus Oculi
135	Davidson	Surgical Instruments: Preparation & Care
141	Dixon & I. Jones	Intraocular Tumors
149	Fink	Binocular Problems in Routine Refraction
155	Gemeroy	External Diseases
159	Gump	Recurrent Iridocyclitis
166	Jacobson	Electroretinography
187	Leopold	Advances in Opthalmic Therapy
188	Lepard	Eye Diseases Common to Children
197	MacLean	Cataracts: Surgical Management
213	Osher	Diagnostic Approach to Surgical Correction of Squint
225	Rychener	The Lacrimal Apparatus
234	Stauffer	Allergic Conditions of Eye
243	Wendland	Chiasmal Lesions

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Tuesday

Key T-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
105	Alvaro	Uveitis: Etiology & Treatment
107	Andrew	Eye Diseases in Children
109	Atkinson	Cataract Extraction
119	Calhoun	Epithelization of Anterior Chamber
121	Cassady	Dacryocystitis & Lacrimal Drainage
132	Cusick	Ptosis Surgery: Practical Aspects
133	Cutler	Cataract: Methods of Extraction
138	DeVoe	Corneal Dystrophies & Degeneration
144	Egan	Cross Cylinder: Application & Theory
153	Fread	Ideal Refraction Equipment
185	Leinfelder	Perimetry & Use of the Tangent Screen
215	Payne	Extraction of Complicated Cataract
223	Ruedemann	Surgery of Vertical Muscles
231	Solanes	Parasitic Uveitis (Metazoal)
236	Swan	Glaucoma: Value of Drugs Used

Key T-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
103	Aldrich	Emotional Problems
112	Berens	Ophthalmic Surgery
142	Dunlap	Divergence Anomalies
146	Falls	Practical Application of Heredity
158	Givner	Eye Changes in Systemic Diseases
168	Jardon	Fitting Ocular Prostheses
178	Kestenbaum	Reading Aids for Low Vision
224	Rundles	Streak Retinoscopy
226	Scarney	Slit Lamp (Biomicroscopy)

Key T-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
113	Berke	Ptosis: Surgical Treatment
115	Bettman	Value of Goniotomy
117	Bonaccolto	Enucleation Techniques
126	Chandler	Cataract Surgery
134	Daily	Biomicroscopy of Vitreous & Fundus Oculi
156	Gibson	Marginal Myotomy
160	Haas	Congenital Glaucoma
172	Joy	Chronic Glaucoma: Surgical Management
177	Kennedy	Ocular Manifestations of Systemic Diseases
184	Leahey	Corneal Transplantation
188	Lepard	Eye Diseases Common to Children
189	Levy	.....SOLD OUT.....
192	Lloyd	Fundus Lesions of the Senile Eye
194	Long	Retinoblastoma & the Pseudoretinoblastomas
206	Morrison	Glaucoma: Office Management
217	Pico	Dacryocystorhinostomy
225	Rychener	The Lacrimal Apparatus
227	Scheie	Congenital Glaucoma: Surgical Procedures
240	Uihlein	Neuralgias of the Head: Treatment
245	Wood	Central Retinal Lesions

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Wednesday

Key W-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
121	Cassady	Dacryocystitis & Lacrimal Drainage
124	Chamberlain	Vertical & Horizontal Components in Strabismus
162	Hedges	Neuralgias of the Head: Treatment
164	Hill	Cataract Surgery: Keratome & Scissors Technique
167	Jampolsky	Ocular Divergence: Management
169	Jobe	Information on Ophthalmic Instruments
171	Johnson, L.	Bullous Keratopathy: Treatment
179	King	Preserved Ocular Tissues Used for Transplantation
182	Kronenberg	Practical Ocular Therapeutics
185	Leinfelder	Perimetry & Use of the Tangent Screen
186	Lennon	Acute Inflammatory Glaucoma: Therapy
196	Luce	Wound Healing
198	Mangold	Proper Fitting of Glasses
199	Mann	Hypertensive Vascular Changes in the Fundus
202	McGraw	Contact Lenses
208	Naquin	Dislocation of the Lens
218	Pino	Aids to the Ophthalmologist in Office Practice
219	Prewitt	External Eye Diseases Associated with Allergy
239	Theodore	Ocular Therapeutics
241	Vail	Senile Cataract: Mechanical Factors of Surgery

Key W-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
125	Chamlin	Perimetry
147	Falls	Ocular Geriatrics
170	Johnson, C.	Ptosis
174	Katz, D.	Ophthalmic Anatomy
178	Kestenbaum	Reading Aids for Low Vision
180	Klien	Clinical Pathology of the Retina
183	Kronfeld	Present Trends in Ocular Pharmacology
193	Lloyd	Diseases of the Cornea
195	Lubkin	Psycho-Somatic Aspects of Ophthalmology
203	McGuire	Cataract: Management of the Patient
204	Meyer	Glaucoma: Surgical Treatment
214	Paulson	Lighting for Comfortable & Accurate Seeing
216	Pfeiffer	Roentgenography of the Eye & Orbit
220	Quereau	Practical Physiology of Ocular Movements
229	Simpson	Hemorrhage Into the Vitreous
230	Smith	Fractures of the Orbit
232	Spaeth	Plastic Surgery: Practical Aspects

Key W-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
101	Aebli	End Results of Muscle Surgery
102	Albers	The Iris in Aphakia
108	Asbury	Practical Genetics
123	Castroviejo	Corneal Surgery
140	Dixon	Indirect Ophthalmoscopy
160	Haas	Congenital Glaucoma
163	Henderson	Intracranial Arterial Aneurysms
165	Iliff	Ptosis
173	Kasper	Dacryocystorhinostomy
175	Katz, I. H.	Common Eye Diseases of Dogs & Their Therapy
176	Kearns	Unusual Neurologic Motor Anomalies
201	McDonald	Penetrating Wounds of the Eye
206	Morrison	Glaucoma: Office Management
207	Murrah	Aids for Subnormal Vision
210	Ogle	Stereopsis: Present Concept
227	Scheie	Congenital Glaucoma: Surgical Procedures
228	Shipman	Scleral Resection
235	Steffensen	Senile Macular Degeneration: Current Therapy
238	Tassman	Retinopathy in Diabetes
240	Uihlein	Neuralgias of the Head: Treatment
242	Weisser	Repair of Corneal Injuries
244	Wise	Retinal Vascular Disease
245	Wood	Central Retinal Lesions

SCHEDULE OF INDIVIDUAL COURSES — OPHTHALMOLOGY — Thursday

Key Th-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
116	Block	Office Practice of Ophthalmology
118	Breakey	Rehabilitation in Ophthalmology
130	Cowen	Pool Gonioscopy
143	Edwards	Abnormal Effects of Prescribed Lenses on Extrinsic Muscles
150	Fink	Vertical Muscle Surgery
180	Klien	Clinical Pathology of the Retina
182	Kronenberg	Practical Ocular Therapeutics
190	Lewis	Mechanical Visual Aids
191	Linn	Choice of Glaucoma Operation
202	McGraw	Contact Lenses
204	Meyer	Glaucoma: Surgical Treatment
209	Newell	Refinements in Ophthalmic Surgery
211	Ogle	Binocular Oculomotor Coordination & Imbalances
212	Olson	Iritis, Iridocyclitis, Keratitis
222	Rudolph	Chemotherapy: New Types
233	Spaeth	Complicated Ptosis: Selection of Operation
237	Tait	Telescopic & Magnifying Lenses for the Visually Handicapped
239	Theodore	Ocular Therapeutics

CONTINUOUS COURSES AND PANEL DISCUSSIONS
OTOLARYNGOLOGY

COURSE	SPEAKERS	SUBJECT	PERIODS USED
301	Brewer & Briess	Voice Therapy	2 PERIODS Th-1, Th-2
302 or 303	Conley, Daly & Lenz	Carcinoma of Head & Neck	2 PERIODS W-1, W-2 or Th-1, Th-2
304	Converse	Nasal Plastic Surgery—Basic	2 PERIODS W-2, W-3
305	Converse	Nasal Plastic Surgery—Advanced	2 PERIODS Th-2, Th-3
306	Fox & Glorig	Noise in Industry	2 PERIODS T-1, T-2
307 or 308	Froeschels	Speech & Voice Therapy	2 PERIODS T-2, T-3 or W-1, W-2
309	Godlowski	I. Physiopathology of Endocrine System II. Enzymatic Mechanism of Allergy	2 PERIODS T-1, T-2
310	Godlowski	Endocrinopathies	2 PERIODS W-1, W-2
311	Guilford & Draper	.....SOLD OUT.....	
312 or 313	Hansel	Allergy & Medical Otolaryngology	2 PERIODS W-1, W-2 or Th-1, Th-2
314	Harbert	Clinical Audiology	2 PERIODS T-1, T-2
315 or 316	Hardy & Pauls	Clinical Audiology for Young Children	2 PERIODS T-1, T-2 or W-2, W-3
317	Hilding	Microanatomy of the Cochlea	2 PERIODS W-1, W-2
318	Hinderer & Philpott	External Pyramid Reconstruction	2 PERIODS Th-1, Th-2
319	House, H., & House, W.	Surgery for Chronic Ear Disease & the Concept of Tympanoplasty	2 PERIODS Th-1, Th-2
320	Kazanjian et al.	Traumatic Injuries of Face	2 PERIODS T-2, T-3
321 or 322	Lathrop et al.	Otological Problems	2 PERIODS T-1, T-2 or W-1, W-2
323	Lindsay	Deafness & Dizziness	2 PERIODS Th-2, Th-3
324 or 325	Mueller et al.	What the Otologist Should Know About Hearing Aids	2 PERIODS T-2, T-3 or W-2, W-3
326	Riggs & Sputh	External Pyramid Reconstruction	2 PERIODS Th-2, Th-3
327	Semenov	Ear, Nose & Throat Therapeutics	2 PERIODS T-1, T-2
328	Senturia et al.	Tubo-Tympano-Mastoiditis	2 PERIODS W-2, W-3
329	von Leden & Moore	Applied Laryngeal Physiology	2 PERIODS Th-2, Th-3
330 or 331	Walsh	What Can Be Done for Deafness	2 PERIODS W-1, W-2 or Th-1, Th-2
332	Williams & Aagesen	Septolobular Considerations in Nasal Surgery	2 PERIODS W-2, W-3
333 or 334	Work	Techniques in Wide Field Laryngectomy	2 PERIODS T-2, T-3 or Th-2, Th-3

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Tuesday

Key T-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
405	Armstrong	Vinyl Plastic Tubing in Treatment of Otitis Media
411	Bolotow	Rhinoplasty: Recent Advances
412	Bordley	Deafness in Infant & Pre-school Child: Etiology
420	Cody	Eustachian Tube
421	Compere	Radiologic Examination of Mastoid, Middle Ear & Eustachian Tube
427	Farrior	Stapes Operability in 3-D
429	Fitz-Hugh	Salivary Glands: Common Disorders
432	Furstenberg	Nasal Obstruction: Management
433	Gardner	Practical Voice Therapy
439	Grandon	Flaps & Grafts in Head & Neck Surgery
441	Hale	Fractures of the Jaws: Diagnosis & Treatment
447	Hilger	Office Diagnostic Methods & Treatments
450	Holinger & Johnston	Endoscopic Aspects of Esophageal Disease
457	Jones	Seasonal Hay Fever: Diagnosis & Treatment
462	Kelemen	Congenital Deafness
466	Kirchner	Facial Bone Injuries
476	Lubart	Sinusitis & Eye Disease
482	McNally	Labyrinthine Tests
512	Sullivan & Smith	Facial Palsies & Their Treatment
513	Tabb	Maxillectomy: Surgical Technique
515	Tremble	Practical Therapeutics in Office Practice

Key T-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
406	Arnold	Destructive Lesions of the Temporal Bone
409	Becker	Plastic Surgery for Protruding Ears
430	Folbre	Discarding Old Ideas in Otolaryngology
446	Henner	Functional Surgery of Middle Ear: Principles
449	Hilsinger	Vascular Headaches
454	Huffman	Reconstruction of Nasal Septum
464	Kemler	Diagnostic & Therapeutic Aid in ENT Practice
474	Loch	Septum Deviations
477	Lynch	Suspension Laryngoscopy & Intralaryngeal Surgery
486	O'Neil	Aerosol Therapy in Otolaryngology
500	Ronis	Chronic Sinusitis: Diagnosis & Treatment
509	Silverman	Education & Rehabilitation of Hearing Handicapped
517	Wallner	Diseases of Larynx: Kodachrome Clinic
519	Weaver	Tumors of the Face: Management

Key T-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
407	Atkins	Diseases of Trachea: Diagnosis & Management
410	Bellucci	Surgery of the Stapes & Oval Window
417	Canfield	Speech Audiometry
422	Corgill	Radical Hypopharyngeal Surgery
424	DeWeese	Problem of the Dizzy Patient
431	Fowler	Facial Paralysis & Tics
437	Goodhill	Stapes Mobilization: Peribasal Monitored Technique
438	Gorney	Esophageal Strictures: Prevention & Treatment
445	Hart	Nonmalignant Lesions of Chest
448	Hill	Paranasal Sinuses
456	Jesberg	Benign Throat Conditions: Diagnosis & Treatment
465	Kinney	Hearing in Children: Newer Concepts
471	Lepper	Antibiotics in Otolaryngology
478	Magielski	Trauma of Larynx & Trachea
491	Pressman	Primary Repair Following Removal of Facial Tumors
499	Roberts	Little Strokes Observed in ENT Practice
506	Sellers	Office Practice of Otolaryngology
508	Shambaugh	Otosclerosis: Selection of Therapy
510	Skolnik	Head & Neck Surgery: Errors & Safeguards
520	Woodman	Glottic Obstruction

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Wednesday

Key W-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	The Adenoid Problem
414	Brodnitz	Actor & Singer in ENT Practice
416	Buckingham	A Clinic in Otoscopic Diagnosis
417	Canfield	Speech Audiometry
418	Cinelli	Physiologic Rhinoplastic Surgery
419	Clerf	Causes of Hoarseness: Treatment
427	Farrior	Stapes Operability in 3-D
436	Goodale	Rationale of Surgery of Frontal Sinus
443	Harkins	Routine Office Management of Allergic Patients
447	Hilger	Office Diagnostic Methods & Treatments
458	Jones	Acute External Otitis: Diagnosis & Treatment
461	Juers	Preservation of Hearing in Surgery
463	Kelemen	.....SOLD OUT.....
472	Lewis	Reactions to Drugs & Antibiotics
480	McBean	Lesions of Nasopharynx Associated with Hearing Loss
482	McNally	Labyrinthine Tests
484	Myers	Surgery of Chronic Otitis Media and Mastoiditis
494	Rabbett & Bellucci	Submucous Resection of Nasal Septum
514	Tabb	Surgical Approaches to Nasopharynx

Key W-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
403	Anderson	Rhinoplasty: Practical Considerations
423	Crispell	Otolaryngologic Allergy in Children
435	Goldman	Correction of Deviated Nose
444	Harrill	Cancer of Maxillary Sinus
454	Huffman	Reconstruction of Nasal Septum
455	Jackson	Peroral Endoscopy & Endoscopic Photography
475	Long	Use of Antibacterial Substances
489	Pearlman	Certain Aspects of Aging in Ear, Nose & Throat Regions
493	Proctor	.....SOLD OUT.....
495	Rawlins	Corticosteroids in Otolaryngology
497	Rinkel	The Nature of Food Allergy
503	Saunders	Treatment & Clinical-Pathologic Correlation of Oral Lesions
511	Steele	Bronchogenic Carcinoma: Diagnostic Aspects
516	Van Alyea	Sinusitis: Modern Trends in Management

Key W-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
408	Baron	Interesting Problems in Otolaryngology: Management
410	Bellucci	Surgery of Stapes & Oval Window
412	Bordley	Deafness in Infant & Preschool Child: Etiology
413	Brewer & Briess	Laryngeal Anatomy & Phonatory Laryngeal Physiology
415	Brown	Cerebellopontine Angle Tumors
424	DeWeese	Problem of the Dizzy Patient
425	Dickinson	Facial Injuries: Basic Problems & Treatment
426	Erich	Fractures of Facial Bones
431	Fowler	Facial Paralysis & Tics
442	Hale	Cysts of Jaws & Mouth: Diagnosis & Treatment
449	Hilsinger	Vascular Headaches
452	Hough	Anatomic Variations in Middle Ear: Significance in Stapes Mobilization
453	House, H.	Stapes Mobilization Procedure: Personal Experiences
459	Jordan, L.	Recent Nasal Fractures: Management
470	Lempert	Temporal Bone Surgery: Pitfalls & Problems
483	Mohs	Chemosurgical Method for Microscopically Controlled Excision of External Cancer with Reference to Cancer of Eyelids
488	Pastore	Lump in the Throat & Pharyngeal Reflexes
492	Pressman	Cysts of the Neck: Diagnosis & Surgical Treatment
504	Schuknecht	Technique for Removal & Preparation of Temporal Bones
507	Sellers	Dizziness: Its Cure or Relief
521	Woodruff	Epistaxis and Its Management

SCHEDULE OF INDIVIDUAL COURSES — OTOLARYNGOLOGY — Thursday

Key Th-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
404	Andrews	Pulmonary Function Studies
419	Clerf	Causes of Hoarseness: Treatment
420	Cody	Eustachian Tube
434	Glorig	The Otolaryngologist & Hearing Aid Selection
435	Goldman	Correction of Deviated Nose
437	Goodhill	Stapes Mobilization: Peribasal Monitored Technique
460	Jordan, R.	Meniere's Disease: Diagnosis & Treatment
461	Juers	Preservation of Hearing in Surgery
468	Lederer	Diagnostic & Therapeutic Pitfalls in ENT Practice
473	Lewy	Self Retaining & Gear-Powered Direct Laryngoscopy
479	Maxwell	Diseases of Salivary Glands: Differential Diagnosis
487	O'Neil	External Otitis: Diagnosis & Treatment
490	Perlman	Fluid in Middle Ear: Etiology & Management
505	Schuknecht	Technique for Using Chisel in Stapes Mobilization
518	Wallner	Operation for Closure of Oroantral Fistula

Key Th-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
409	Becker	Plastic Surgery for Protruding Ears
440	Guilford & Wright	Controlled Healing of Mastoid & Fenestration Cavities
455	Jackson	Peroral Endoscopy & Endoscopic Photography
467	Kos	Stapedial Mobilization
469	Lemon	Serous Otitis Media in Children
481	McLaurin	The Child Without Speech: Differential Diagnosis & Treatment
496	Reger	Practical Office Audiometry
501	Rosen	Surgical Anatomy of Vestibule with Reference to to Stapedial Footplate
502	Ryan	Headache: Differential Diagnosis & Treatment
516	Van Alyea	Sinusitis: Modern Trends in Management

Key Th-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
402	Alfaro	Fullness in the Ear: Differential Diagnosis
408	Baron	Interesting Problems in Otolaryngology: Management
426	Erich	Fractures of Facial Bones
428	Farrior	Tympanoplasty in 3-D
432	Furstenberg	Nasal Obstruction: Management
451	Holinger & Johnston	Benign Tumors of Larynx: Treatment
465	Kinney	Hearing in Children: Newer Concepts
470	Lempert	Temporal Bone Surgery: Pitfalls & Problems
477	Lynch	Suspension Laryngoscopy & Intralaryngeal Surgery
483	Mohs	Chemosurgical Method for Microscopically Controlled Excision of External Cancer with Reference to Cancer of Eyelids
485	Ogura	Radical Neck Dissection & Laryngeal Carcinoma
498	Rinkel	Specific Diagnosis in Allergy
508	Shambaugh	Otosclerosis: Selection of Therapy
515	Tremble	Practical Therapeutics in Office Practice

SECTION ON INSTRUCTION

ADVANCE ORDER SHEET

All courses cost $2.00 for each period (one hour). Follow instructions explicitly and check for errors in listing before mailing.

Orders will not be accepted for presale after October 2. To ensure acceptance they should be mailed before September 29 to

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
15 Second Street S.W., Rochester, Minnesota

Academy Candidates and Guests May Purchase Tickets ONLY at Instruction Desk in Registration Foyer

(Over)

ADVANCE ORDER SHEET

ORDERS FILLED ONLY IF CURRENT DUES ARE PAID

NO REFUNDS WILL BE MADE AFTER OCTOBER 10

(Type or Print) Last Name First Name Middle Initial

Office Address

City Zone State

Days to be Present

- ☐ Monday
- ☐ Tuesday
- ☐ Wednesday
- ☐ Thursday

Total Amount Enclosed $_____

SELECTION OF COURSES ON REVERSE SIDE OF THIS BLANK

RECAPITULATION OF GENERAL INSTRUCTIONS

1. Monday, Tuesday, Wednesday, and Thursday are represented by the letters M, T, W, Th. The first period in the morning is represented by figure 1, second period by figure 2 and third period by figure 3. The first period in the afternoon is represented by figure 4, second period by figure 5, and third period by figure 6. All courses in otolaryngology and maxillofacial surgery are presented in the mornings. All courses in ophthalmology are presented in the afternoons.

2. Reservation of instruction tickets in advance of the meeting will be made ONLY for MEMBERS whose 1958 dues have been paid. ACADEMY CANDIDATES AND GUESTS MAY MAKE SELECTION OF COURSES *ONLY* AT THE GENERAL REGISTRATION DESK IN CHICAGO.

3. Make check payable to W. L. Benedict, M.D.

4. Mail order promptly to ensure reservation of first choices. If necessary, an attempt will be made to rearrange schedule to include "first choices."

(Over)

SELECTION OF COURSES

PERIOD		FIRST CHOICE		SECOND CHOICE		THIRD CHOICE	
		Course No.	Lecturer	Course No.	Lecturer	Course No.	Lecturer
MON.	4						
P.M.	5						
	6						
TUES.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						
WED.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						
THURS.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						

News - - - Notes

CALL FOR PAPERS

The 1959 meeting of the Section on Ophthalmology, American Medical Association, will be held in Atlantic City, June 8 to 12, 1959.

Anyone wishing to present a paper before the Section is urged to communicate with Harold G. Scheie, M.D., Secretary, Section on Ophthalmology, 313 S. 17th St., Philadelphia 3, as soon as possible. A title and abstract of 50 to 150 words must be in his hands by January 1, 1959.

Anyone wishing to present a scientific exhibit should contact Frank W. Newell, M.D., Representative to Scientific Exhibit, 950 E. 59th St., Chicago 37, as soon as possible.

A $250 prize is offered for the best paper and another $250 prize is offered for the best scientific exhibit prepared for the Section on Ophthalmology at each scientific session.

FALL MEETINGS

The American Society of Ophthalmologic and Otolaryngologic Allergy will hold its annual dinner meeting in Private Dining Room 14 at the Palmer House in Chicago, October 16. Come at 6:00 p.m. for cocktails; dinner begins at 7:00 p.m.

The Pan-American Association of Ophthalmology will sponsor Fiesta Panamericana, a cocktail buffet, at the Hotel Sherman in Chicago, October 14. Service will be from 6:00 to 8:00 p.m.

The International Association of Secretaries of Ophthalmological and Otolaryngological Societies will meet September 12, at 9:00 a.m., at the Palais de Beaux Arts, Brussels. Discussions will include the need for more world wide cooperation of societies, encouraging young men to participate in scientific congresses, and methods of increasing cooperation between ophthalmologic societies and otolaryngologic societies.

Dr. Oscar J. Becker of Chicago was elected vice-president of the American Society of Facial Plastic Surgery when the Society met in New York July 16.

The next meeting of the Society will be held on November 12 in conjunction with the Section on Laryngology, Rhinology and Otology of the Medical Society of the County of Kings and Academy of Medicine of Brooklyn, 1313 Bedford Ave., Brooklyn.

SECTION ON OPHTHALMOLOGY, AMERICAN MEDICAL ASSOCIATION

The annual scientific session of the Section on Ophthalmology, American Medical Association, was held in San Francisco June 24 to 26. Dr. Peter C. Kronfeld was awarded the Prize Medal in Ophthalmology for his many contributions to the field.

The $250 prize for the best exhibit was given to Drs. L. K. Garron, M. J. Hogan, W. K. McEwen and M. L. Feeney for their exhibit, "Electron Microscopy of Ocular Tissue." Dr. Warren A. Wilson was awarded the $250 prize for his paper, "Galactosemia with Associated Cataracts in Children," judged on the basis of presentation and originality.

Dr. Frank B. Walsh was elected chairman, Dr. Philip M. Lewis is vice-chairman, Dr. Henry F. Allen is assistant secretary, Ralph O. Rychener is delegate to House of Delegates, with Dr. W. Howard Morrison as his alternate, and Dr. Frank W. Newell is representative to Scientific Exhibit.

POSITIONS AVAILABLE

The officers and directors of the Eye Bank and Sight Conservation Society of Virginia are seeking a BIOCHEMIST, man or woman, with a Master's degree or better, to do research work. The salary is open. For further information contact E. G. Gill, M.D., Director, Eye Bank and Sight Conservation Society of Virginia, 711 S. Jefferson St., P.O. Box 1772, Roanoke, Va.

A Board-qualified OPHTHALMOLOGIST and a Board-qualified OTOLARYNGOLOGIST are wanted to join a 17 man group in new clinic building. This three-college city of 70,000 has a surrounding referral area of 200,000 people. Minnesota lake resorts are adjacent.

For further information contact Dakota Clinic, 1702 Thirteenth St. South, Fargo, N. D.

A young, well-trained OPHTHALMOLOGIST is wanted to gradually take over the active practice of retiring physician. For further information contact A. E. Jackson, M.D., 3204 S. University Dr., Fort Worth, Texas.

A second-year residency in OPHTHALMOLOGY is available immediately at Veterans Administration 500-bed teaching hospital affiliated with Washington and St. Louis universities. The applicant must be a citizen of the United States or a noncitizen who is a graduate of a school of medicine approved by the American Medical Association. For further information write Personnel Officer, VA Hospital, 915 N. Grand, St. Louis 6.

The Eye Bank and Sight Conservation Society of Virginia has an opening for a PHYSICIAN, who has had two years in OPHTHALMOLOGY and wants to spend one year in research in diseases of the corneas, keratoplasty, toxoplasmosis, tonography and eye clinic work. For further information contact E. G. Gill, M.D., Director, Eye Bank and Sight Conservation Society of Virginia, 711 S. Jefferson St., P.O. Box 1772, Roanoke, Va.

An OTOLARYNGOLOGIST, Board qualified or certified, is wanted to join a 20-man group of specialists in a clinic in a Midwest college town. A man with plastic and endoscopy training is preferred but this training is not a requisite. One Board-certified otolaryngologist is already in the group. Starting salary open; a partnership is possible in two years. For further information contact Byrl D. Houck, Business Manager, McFarland Clinic, Ames, Iowa.

A young Board-certified or eligible OTOLARYNGOLOGIST is wanted to join the staff of the Geisinger Memorial Hospital and Foss Clinic. The department, which is growing rapidly, at present has two staff men and three residents. For further information please write to F. W. Davison, M.D., Director, Department of Otolaryngology and Broncho-Esophagology, Geisinger Hospital, Danville, Pa.

OTOLARYNGOLOGIST needed in Claremont, Calif. Private practice with clinic potential. Write to Morrill L. Ilsley, M.D., P.O. Box 24, Claremont, Calif.

Listings of positions available should be sent to W. L. Benedict, M.D., 15 Second Street S. W., Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist: diplomate, resident, etc.) (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published only once.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse nor guarantee any of the published listings.

FOREIGN SUBSCRIPTIONS

Some foreign members of the Academy as well as subscribers to the TRANSACTIONS are experiencing difficulty in remitting in American dollars. For their convenience the Academy will accept any legal foreign currency in payment of dues, fees for Home Study Courses, Teachers' Manuals, Orthoptic Journals, TRANSACTIONS, etc., at the prevailing rate of currency exchange.

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